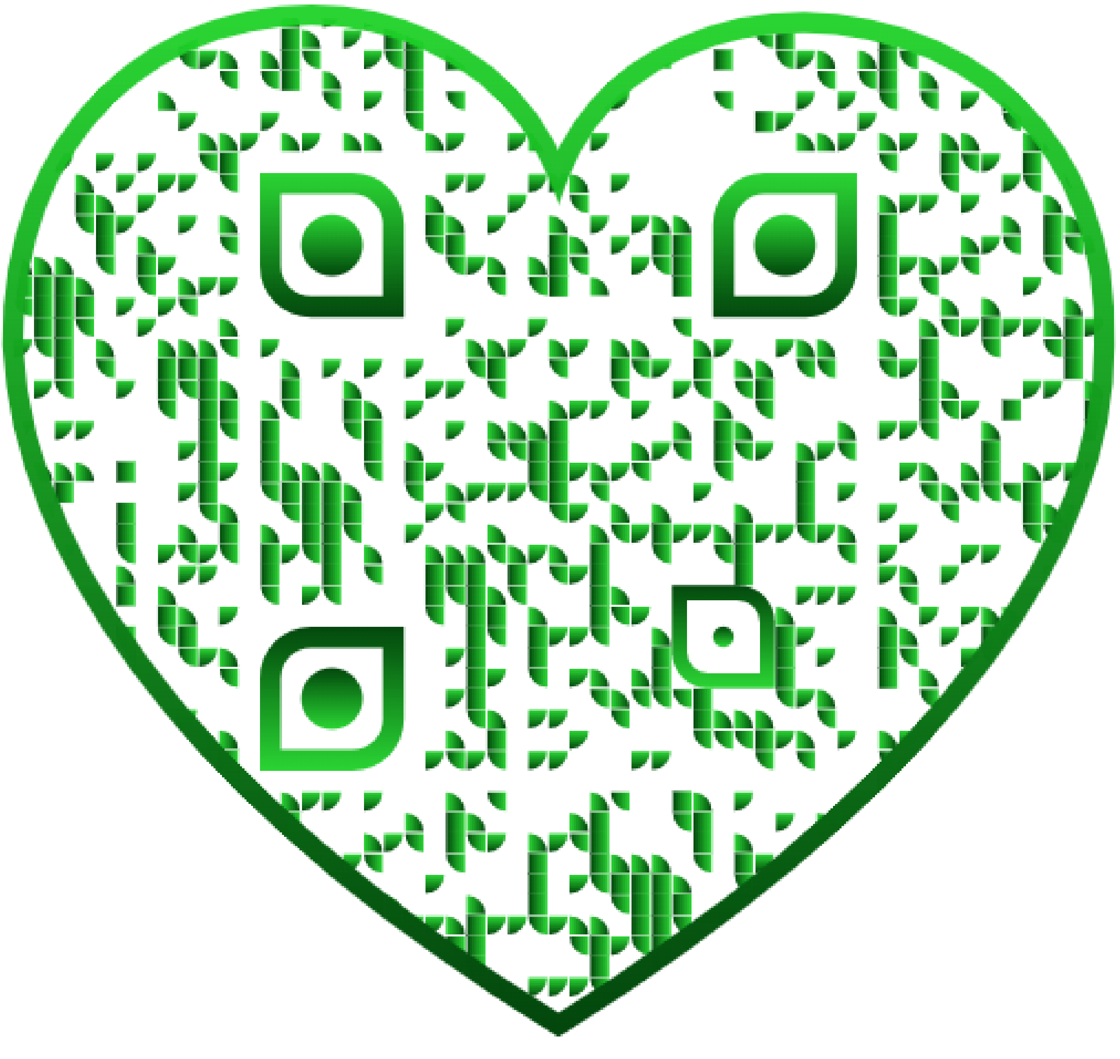
Clinical toxicology

علم السموم السريرية

البروفيسور الدكتور

نبيل حزام الحمادي



General treatment of poisoning

1.Hospitalization: A. B. C., and to prevent further administration of poison.

2.Decontamination of the G.I.T:

In cases of ingested poisons:

a. Induction of vomiting:

- **mechanical**
- **syrup Ipicac (15 – 30 cc)**
- **central emetic,Apomorphine (6 mg S.C)**

b. Stomach wash (gastric lavage).

c. Purgatives.

3. Antidotes: used when:

1. **The poison has not been removed by emesis or gastric lavage.**
2. **The poison is already absorbed.**
3. **The poison has been adminsterd by a root other than ingestion.**

4. Help excretion of poison: by purgatives and diuretics.

5. Symptomatic treatment.

Active Elimination of Poisons

1. Forced diuresis:
   a. Foreced alkaline diuresis.
   b. Foreced acid diuresis.
2. Haemodialysis.
3. Haemoperfusion.

I. Forced diuresis: altering urine PH by:

- Acidification: quinine, amphetamines
- Alkalinisation: salicylates, phenobarbitohne

Using:

- Isotonic sodium bicarbonate solution 1.26%
- 5% dextrose
- 0.9% saline
- kcl-

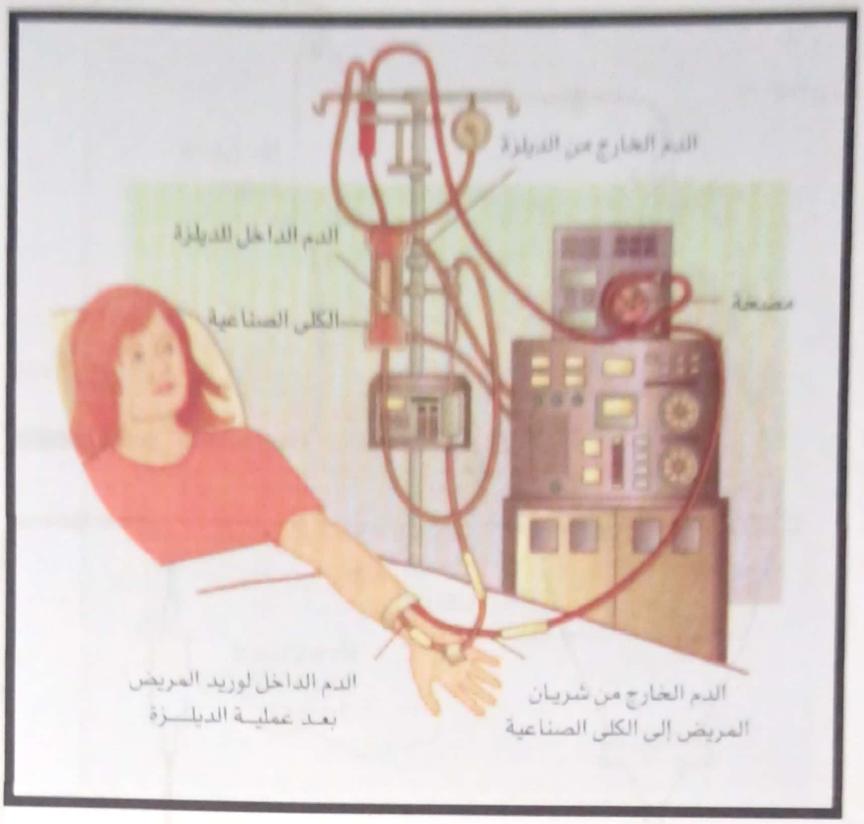
Contraindicated in heart and renal diseases

II. Haemodialysis:

Eliminating small molecules (MWt 350 Daltons)

Maximum removal of the poison depends on:

1. Plasma concentration of poison (diffusion gradient).
2. Protein and tissue binding (free drug).
3. Water solubility of the poison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الدم الداخل لوريد المريض بعد عملية الديلزة
الدم الخارج من شريان المريض إلى الكلى الصناعية

الغسيل الكلوي

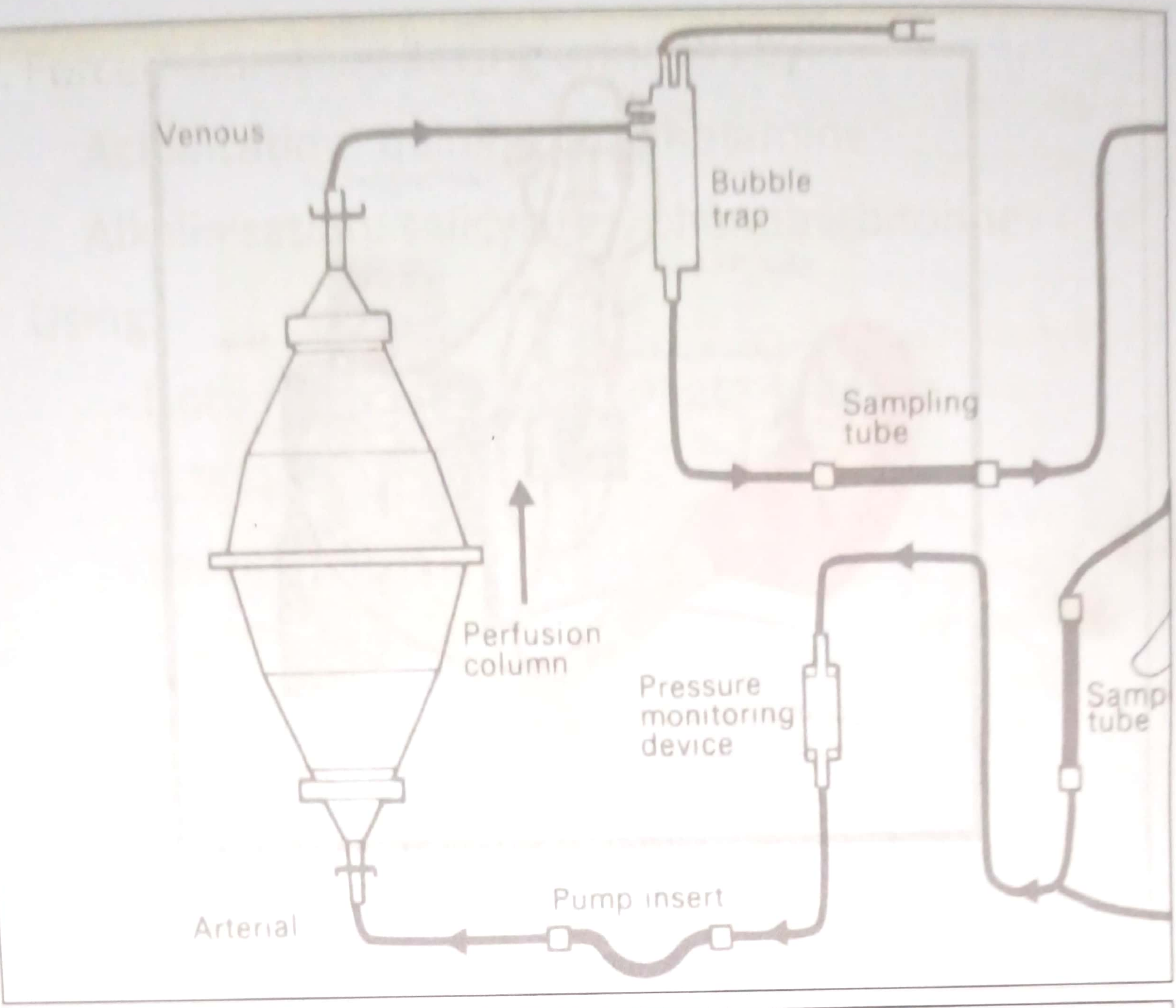
Haemodialysis

III. Haemoperfusion:

Useful in: Hypnotics, gltithemide, barbiturates.

Complications:

- **Charcoal embolisation**
- **Hypotension**
- **Thrombocytopenia**
- **Leucopenia**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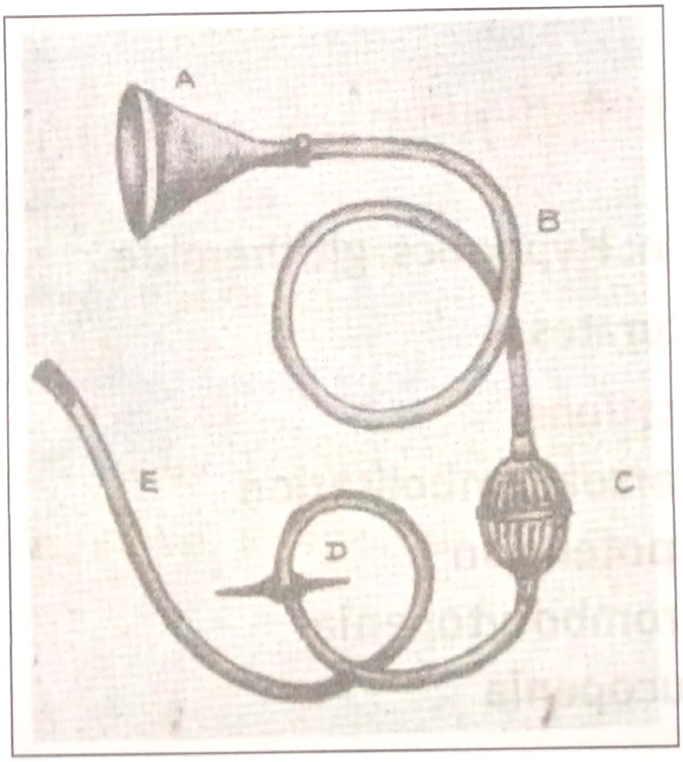
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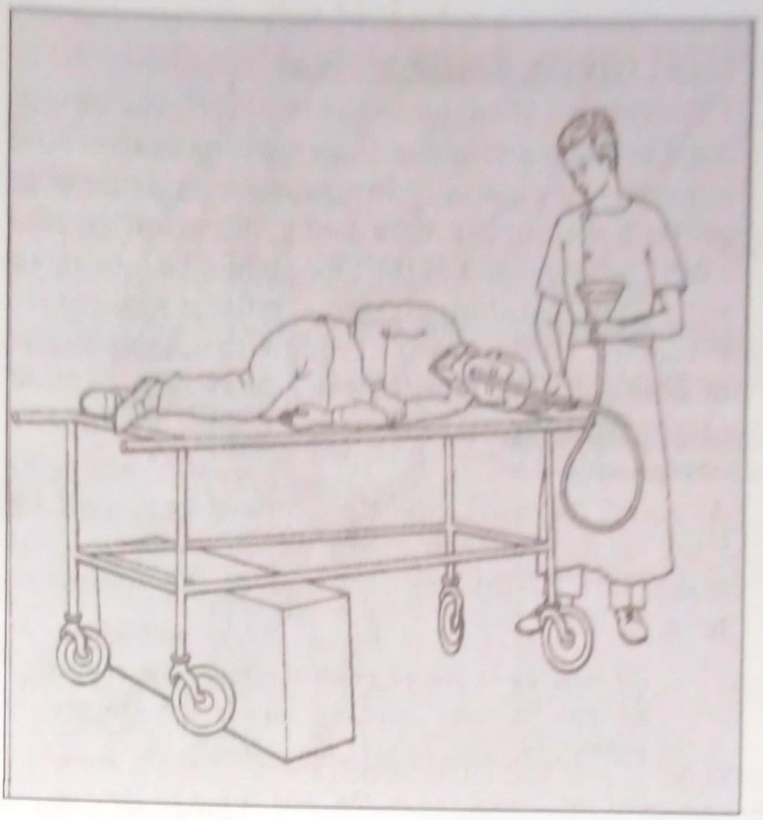

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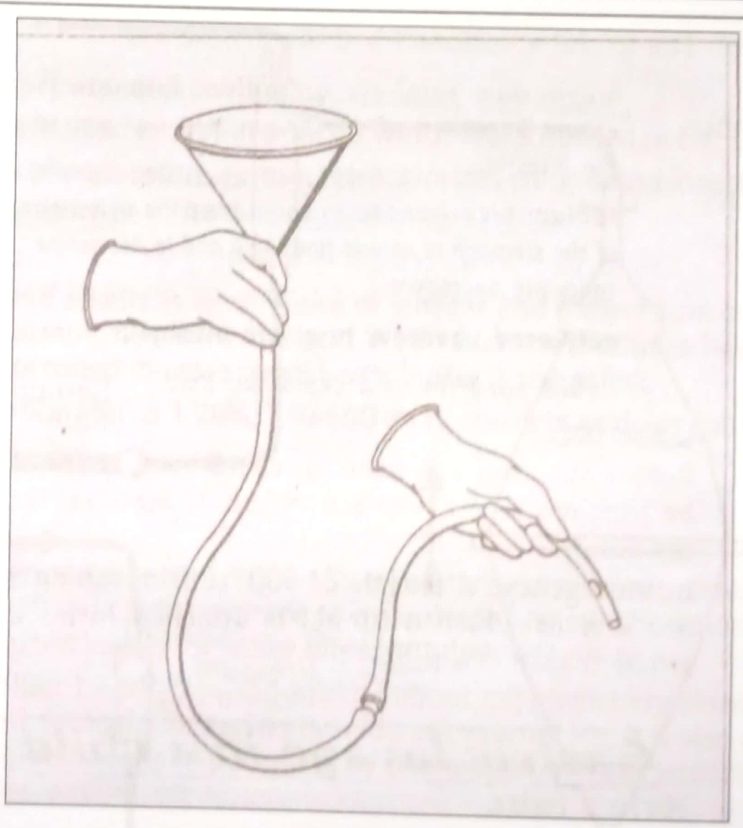

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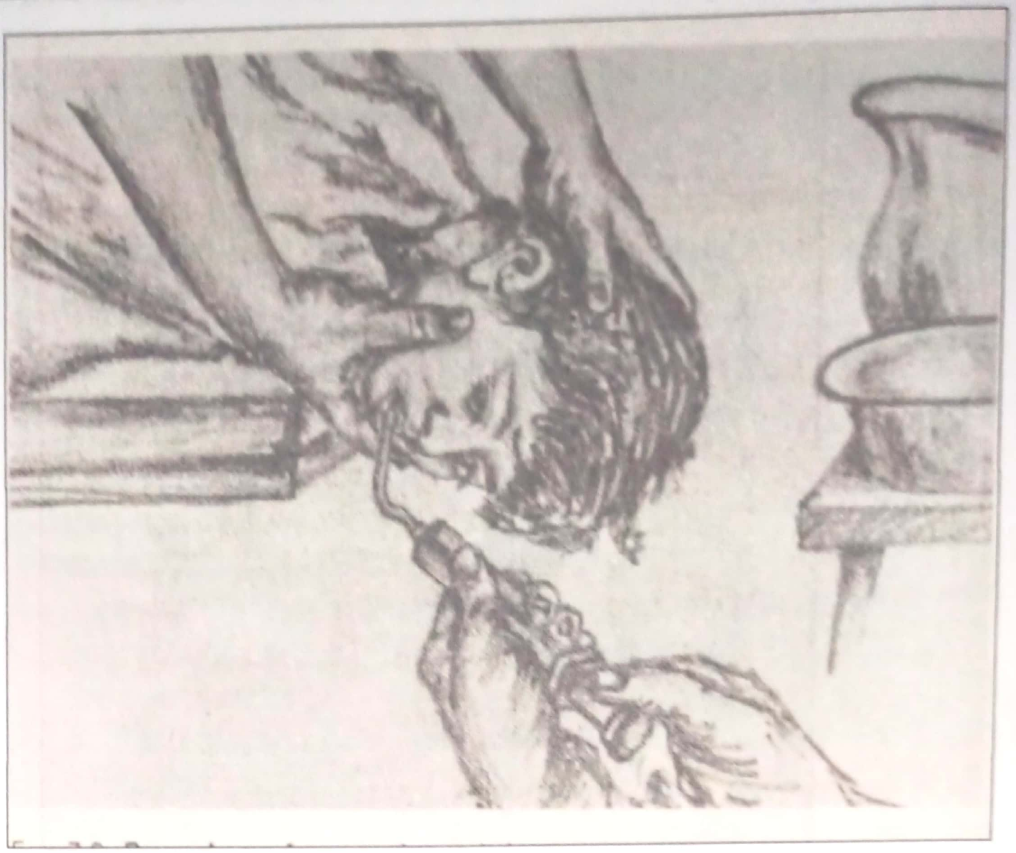

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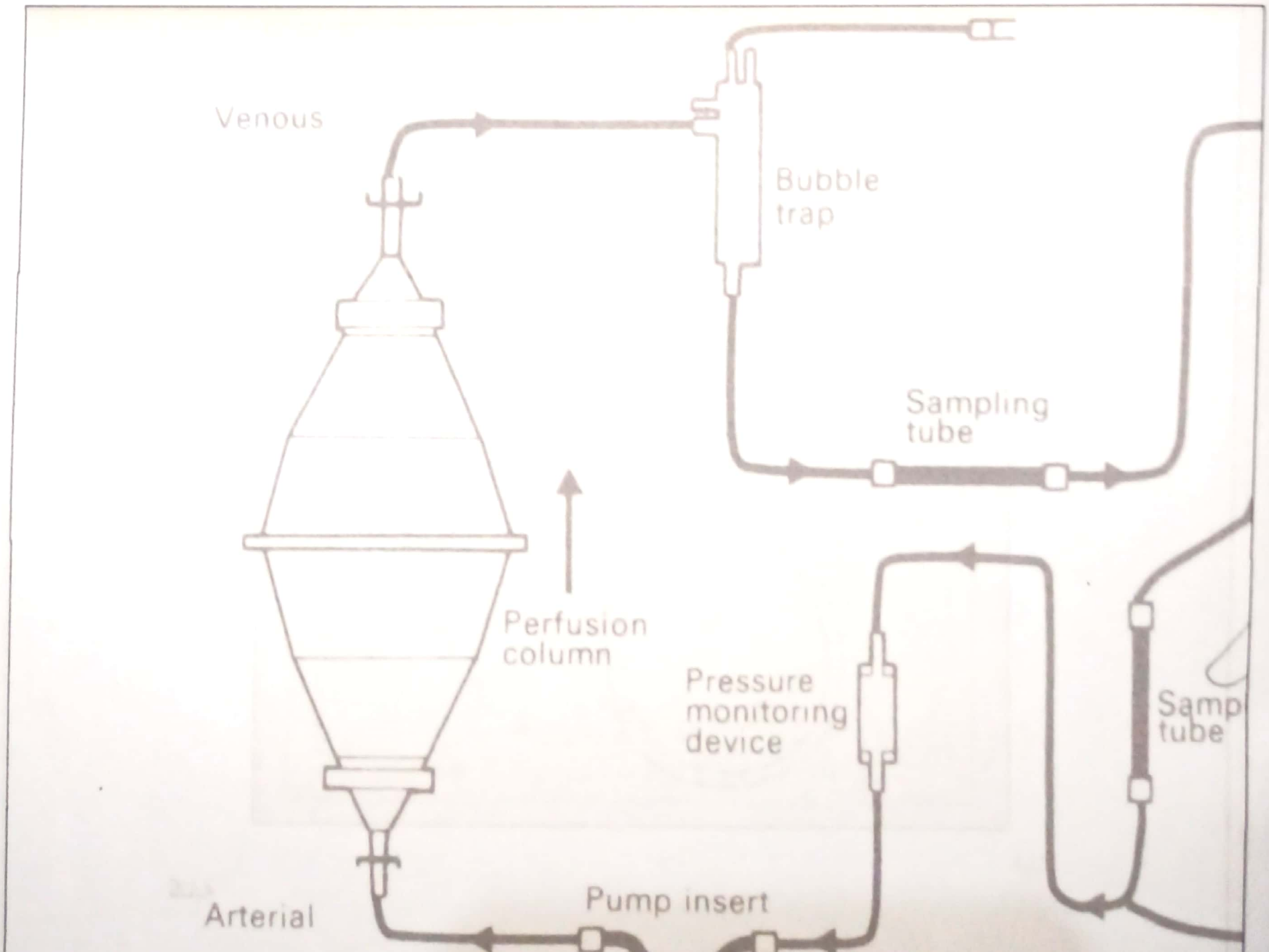
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tube
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Classification of Narcotics (Drugs of abuse=illicit drugs) according to WHO
&
Controlled Drugs

1. Opium & it's derivatives(morphine, heroin, pethidine, tramadol .... etc
2. Cocaine
3. Alcohol
4. Benzodiazepines
5. Barbiturates
6. Antidepressants
7. Stimulants (amphetamine, khat)
8. Hallucinogens (Hashish, LSD, Datura , nutmeg ... etc)
9. Solvents (glue, paint, benzene ,acetone .... Etc)

Chemical classification of Narcotics:

1. Opium :

Opium: Is the brown dried latex of the poppy capsule (papaver sominiferum)

2. Opiates: Normally occurring alkaloids in opium:

- –Morphine 10% of opium, is the most active alkaloid in opium.
- –Nascapine.
- Papaverine 1% of opium, it is not addictive, it is a vasodilator.

– Baine (baine and papverine are not analgesics).

– Codeine (Methyl morphine) 0.5%

3.Opiates derivatives:

– Heroin (Diacetyl morphine) is a very strong addicting agent.

– Dicodid (Dihydrocodeine).

– Nalorphine is used as an antidote for acute opium poisoning.

- Apomorphine has no narcotic effect

4. Synthetic Narcotics:

- **Pethidine** which is a strong anaelgesic antispasmodic with an addictive liability.
- **Methadone** is a synthetic narcotic, it differs than other narcotics in having a prolonged withdrawal state longer than other narcotics but of a lesser intensity, that's why is used as a substitution therapy for other narcotics.
- **Dextropropoxyphene** has a good analgesic potency without a measurable addiction liability.
- **Dionine** (Ethyl morphine), produces conjunctival hyperemia and thus may be used for the treatment of corneal ulcer .

Clinical features in acute opium poisoning:

- Cortical depression Drowsiness Sleep Stupor Deep coma.
- Sweating.
- Pin point pupils (due to stimulation of pupillo-constrictior center).
- Slow full pulse.
- Smell of opium in patient's breath (opium nauseating smell), no smell in case of morphine.
- Nausea and vomiting.
- Skin : cyanotic and covered with cold sweat.
- Respiratory center depression cyanosis decrease breathing death.

Fatal dose:

- opium 2 gms.
- morphine 200 mg addicts can tolerate much bigger doses, children are very susceptible to the action of morphine and only small doses even 1 mg /kg may be fatal to them.

Fatal period: 6-12 hours.

Cause of death:

Central asphyxia.

Differential diagnosis:

- *Coma and pin point pupils is also seen in:*
    - Oragnophosphorous poisoning but with a characteristic increase of all body secretions, particularly increase bronchial secretions, smell of garlic odor.
    - Pontine hemorrhage which is characterized by hyperpyrexia , paralytic signs, extrapyramidal signs.
    - Acute ethanol poisoning which is characterized by pin point pupils that dilate on stimulation of the muscles of the face. McEwin"s sign.
- Toxicological analysis can prove the presence of opium.

Uses of opium & other Narcotics products:

1. Pain killers: morphine, pethidine, tramadol
2. Anesthesia:Xylocaine, lidocaine, lignocaine
3. Cough suppressors: Codeine , dextromethorphan
4. Emetics: Apomorphine
6. Substitution therapy in addiction:
   Methadone,codeine.
7. Treatment of acute opium poisoning: Nalorphine

ADDICTION

الادمان

Addiction:

Is a state of chronic poisoning due to repeated use.

characters of addiction:

1. Tolerance
2. Physical dependence
3. Withdraual symptoms

Treatment of Addiction

1. Admission to a special center to prevent further administration of the agent as well.
2. Gradual withdrawal:
   For opium, benzodiazepines, barbiturates, antidepressants
   Sudden withdrawal for Alcohol and Cocaine.
3. Subistitution therapy with methadone.
4. Good nutrition
5. Religious support.
6. Supportive treatment using the followings:
   Use sedatives only when necessary (preferably clonazepam, other stronger agents like carbamazepine, amitriptyline should be only used for severe withdrawal effects.

AMPHETAMINE

Amphetamines are a group of drugs having sympathomimetic effects but they have more central effect and less cardiovascular effect.

General Uses:

- Induce euphoria
- Decrease fatigue
- Anorexogenic
- Induce alertness

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Captagon كبتاجون
www.alwahm.net
alwahm.net

20 Comprimés
20 Comprimidos

Therapeutic uses:

- Attention deficit hyperactivity in children (no addiction).
- Parkinsonism (it releases dopamine in basal ganglia).
- Chronic alcoholism
- Psychic disorders.
- Nocturnal enuresis
- Fatigue physical and mental.
- Obesity
- narcolepsy

Route of intake:

Inhalation, injection & ingestion.

Preparations:

- Amphetamine sulfate (Benzedrine).
- Dextroamphetamine sulfate (Dexedrine)(Maxton Forte).
- Endroline, has more muscle analeptic effect mainly used in athletes
- Captagon is the most recent form of amphetamine(in drug abuse market).

Mechanism of action:

Indirect symapthomimetic (release of Noradrenaline):

- Stimulation of cerebral cortex producing psychic effects.
- Stimulation of reticular activating system causing analeptic effect.
- Suppression of appetite producing anorexia (depression of satiety centre in hypothalamus).

Fatal dose and fatal period:

Less than 200 mg, death has followed 5 days use of more than 140 mg.

Picture of Poisoning:

- Euphoria
- Mood elevation
- wakefullness.

In toxic doses causes:

- Excitation, insomnia, hyper-reflexia, dilated reactive pupils, hallucination (auditory and visual).
- palpitations.

- GIT symptoms (nausea, vomiting, abdominal pain and diarrhea).
- Convulsions and deep coma are characteristics of severe poisoning.

Treatment:

- Gastric wash by water and activated charcoal.
- Sedation by chlorpromazine
- Acidification of urine by ammonium chloride orally or IV to increase excretion.
- Symptomatic treatment: control palpations, blood pressure and convulsions.

LOVE
125 mg

Designer drugs

- Designer drugs are those drugs having funny symbols (to facilitate the conduction of such drugs of abuse escaping the legal responsibility).

Examples:

- Amphetamines:"Love pill","Love drug", "Ecstasy", "Eva", "Eve", The amino-group can be methylated and then it yields a designed methamphetamine. By addition of methoxyl-goups it yields:

- Methylenedioxyamphetamine (MDA) "Love pill or Love drug, or speed for lovers".
- Methylenedioxyethylamphetamine (MDEA) or MDE) " Eva or Eve".
- Bromodimethoxyamphetamine (DOB) "Bolden eagle", which is 100 times stronger than the previous three amphetamine types.

- NOTE: DOBY drugs are those taken during sport matches and strictly prohibited and punishable on use by law.

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BENZODIAZEPINES

They belong to the group of the anxiolytic sedatives (minor tranquillizers) reduce pathological anxiety, tension and agitation. In higher doses they have anticonvulsant, myotonolytic and narcotic effects. They may cause dependence and addiction already after two to four weeks (low dose dependence).

Therapeutically they are divided into 3 groups:

Group1:

- Substances which are in the original state ineffective and act by the metabolites produced from the organism; e.g. Chlordiazepoxide (Librium), the first successful benzodiazepine, developed in the late 1950s. To this group belong Clorazepate, prazepam, medazepam and flurazepam.

Group 2:

- These substances are themselves effective and therefore show a rapid effect. Their metabolites are also effective and are more slowly eliminated than the original substance. To this group belong diazepam (valium, valinil), flunitrazepam.

Group3:

They are effective in their original form and are rapidly metabolized. They are used for short acting effects.

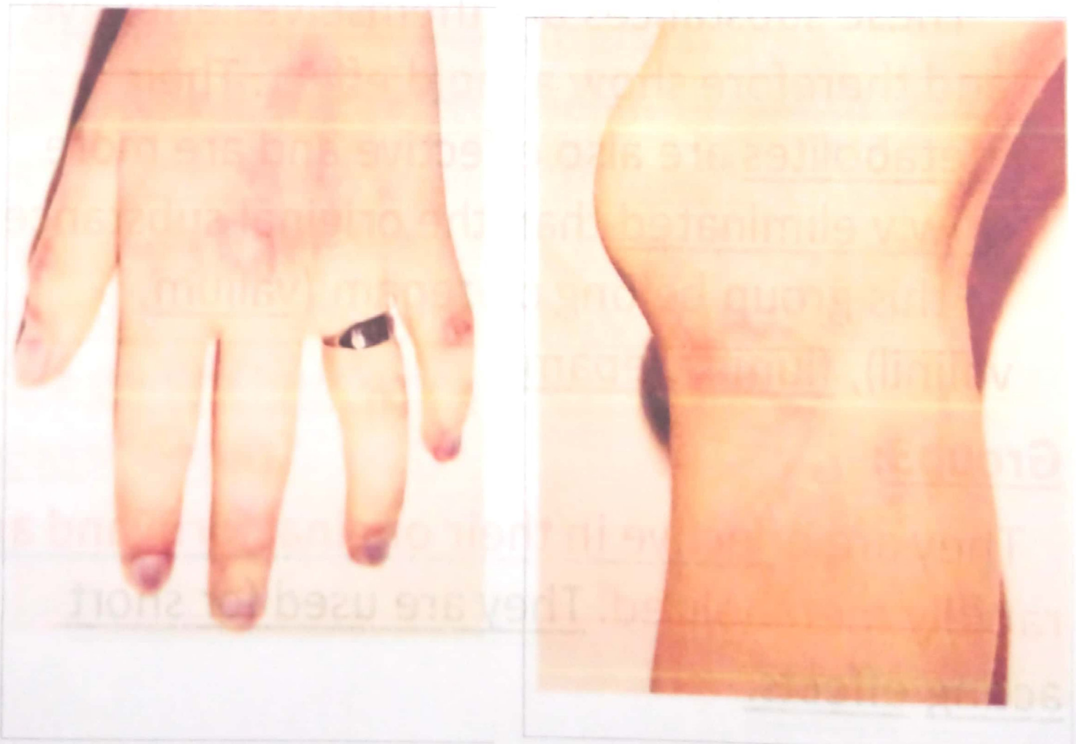
e.g. sleep or narcosis induction. Examples of this group are triazulam, brotizolam, midazolam, oxazepam, bromazepam, nitrazepam. Alprazolam (restyl or alpranex).

Clinical Picture:

- Drowsiness
- Dysartheria
- Partial ptosis
- Hallucinations
- Nausea, headache and vertigo
- Bullous skin lesions
- Hypotension

Overdose by benzodiazepine

محاولة الانتحار



Chronic poisoning:

- Depression
- Ataxia
- Increase appetite
- Impairment of libido

Treatment:

1. Stomach wash by activated charcoal
2. The specific antidote is **flumazenil** I.V 0.2 mg every 60 seconds, further 0.1 mg is given until 1 mg is reached.
3. Care of respiration.

TRICYCLIC ANTIDEPRESSANTS

These represent the largest group of drugs used for the treatment of depression.

Uses:

1. Treatment of depression as they cause mood elevation (in normal persons they cause sedation).
2. Treatment of enuresis.

Mode of poisoning:

a. accidental b. suicidal through oral route.

Action:

Decrease the action of acetylcholine both centrally and peripherally.

Preparations:

- Amitryptyline (Tryptizol)
- Imipramine (Tofranil)
- Doxepin (Sinequan)

New tricyclic antidepressants

These new antidepressants have become the most commonly used antidepressants replacing the old TCAs as first line of depression treatment due to their selective action and less side effects.

They include specific serotonin reuptake inhibitors (SSRIs):

1. Fluoxetine (Prozac)
2. Sertraline
3. Paroxetine
4. Fluvoxamine

Clinical Picture:

Initial symptoms are those of C.N.S depression:

- **ataxia, agitation, disorientation**
- **Respiratory depression**
- **Seizures**
- **Pyramidal signs**
- **Anticholinergic (atropine) effects**
- **C.V.S effects are the most dangerous:**

  Cardiac arrhythmias, ventricular rate is more than 120 beats/min, hypotension, and cardiac arrest.

Treatment:

- Emesis and gastric lavage using charcoal and saline cathartics
- Physostigmine 1-2 mg I.V.

Hypnotics

Barbiturates

Classification:

1. Long acting: 8-10 hours e.g. phenobarbitone (luminal).

2.Intermediate acting: 5-6 hours

e.g. pentobarbitone (nembural) or amytal.

3.Short acting: 2-3 hours

e.g. hexobarbitone (evipan) or seconal.

4.Ultra short acting: used as anaesthetic agents e.g. Thiopenton sodium (pentothal).

Uses:

- Hypnotics.
- Sedatives.
- Antiepileptics.
- Anaesthetics.
- Psycho-analysis: ultra short barbiturates.

Mode of poisoning:

- Suicidal: common because they are easily obtained and they lead to coma followed by painless death.
- Accidental: during their therapeutic use.
- Homicidal: rare because of large fatal dose.

Fatal dose:

- Long acting: about 4 gms of luminal
- Intermediate acting: 1.5-2 gms
- Short acting: 1-1.5 gms
- Ultra short acting: 1 gm

Barbiturates and alcohol have synergistic effects

Clinical features:

1. CNS depression: cortical depression, deep sleep, coma → respiratory depression.

2. Barbiturate coma: is accompaned with:

- Cyanosis.
- Slightly dilated reactive pupils.
- Absent reflexes.
- Stertorous breathing.
- Rapid weak pulse.
- Decrease B.P.
- Albuminurea and haematurea.
- Skin may show areas of erythema.

Treatment:

1. Stomach wash: cuffed endotracheal tube should be used to prevent aspiration if the patient is in coma.
   Wash with potassium permanganate 1/5000 and leave a purge in the stomach
2. Care of respiration.
3. Catheterization for urine retention.
4. Prophylactic antibiotics.

Causes of death:

- Central asphyxia due to depression of respiratory center.
- Hypotension.
- Pneumonia.

بروفيسور.دكتور/نبيل حزام الحمادي

أستاذ الطب الشرعي والسموم
رئيس قسم الطب الشرعي والسموم
كلية الطب – جامعة صنعاء

استشاري الطب الشرعي لدى المحاكم والنيابات

عضو هئية التدريس بالمعهد العالي للقضاء
رئيس لجنة الخبراء
وزارة العدل

CORROSIVE POISONS

Definition: any substance that in contact with living tissue will cause destruction by it's chemical action.

Classification:

1-Inorganic acids: Sulfuric, nitric and Hcl acids.

2-Caustic alkali: Na hydroxide, K+ hydroxide (caustic potash) and ammonium hydroxide.

3-Organic acids as oxalic, carbolic and acetic acids.

4-Metal corrosives (mineral salts): Mercuric chloride and antimony chloride…etc.

USES OF CORROSIVES:

1.ACIDS:

Hydrochloric acid: used in

- swimming pool cleaners.
- Toilet bowl cleaners (Flash or Harbic).
- Dye manufacturing.

Sulfuric acid: used in:

- Battery acid
- Toilet bowl cleaners.
- Wine fermentation.

Nitric acid: used in:

- In electroplating.
- Fertilizer manufacturing.

2. ALKALIS:

Na and potassium hydroxide used in:

-Oven cleaners

-Detergents.

-Paint removers

-Washing powders

Ammonium hydroxide: used in:

-Detergents

-Bleaching agents

-Jewelry cleaners

3. OTHERS:

Clorox liquid (Na hypochlorite)

- Swimming pool sanitizers.
- Bleaching agents.

Other uses of corrosives:

- Explosives
- Button batteries (alkaline or acid batteries)

Clinical picture of corrosive poisoning

1 - Severe burning pain from the mouth down to the stomach.

2- Eschars.

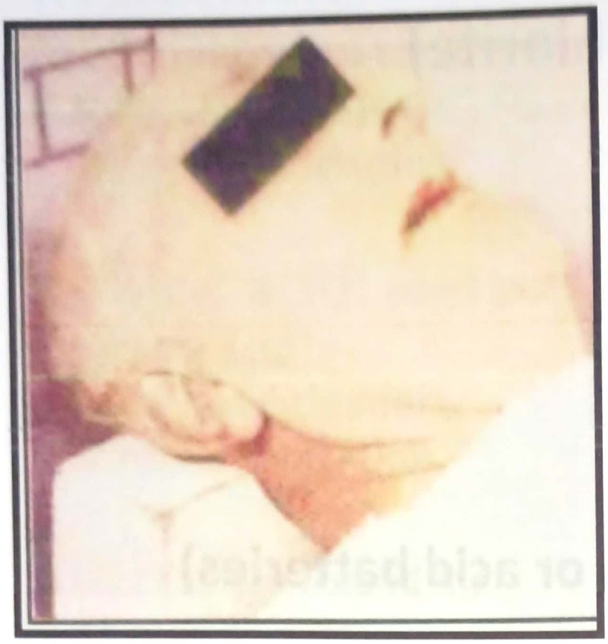
3- Vomiting & diarrhea.

4- Clothes may show streaks of charring.

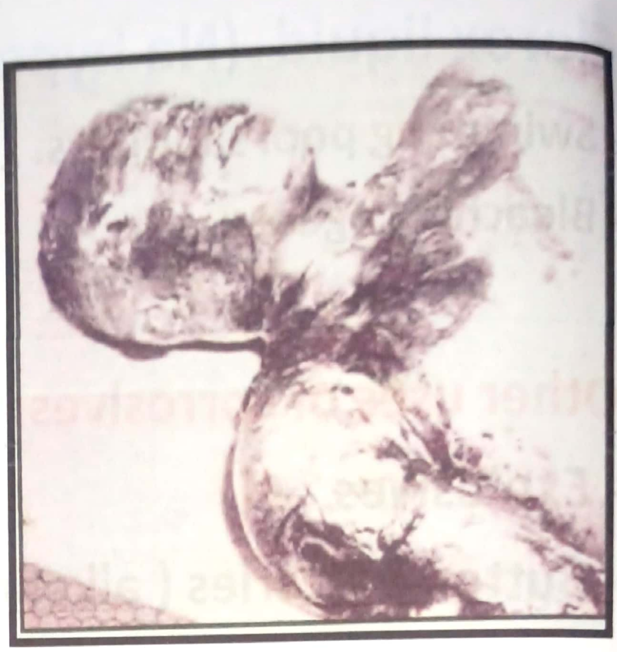
5- Pulmonary complications e.g. pneumonia, edema glottis.

6- Skin &Corneal corrosions.

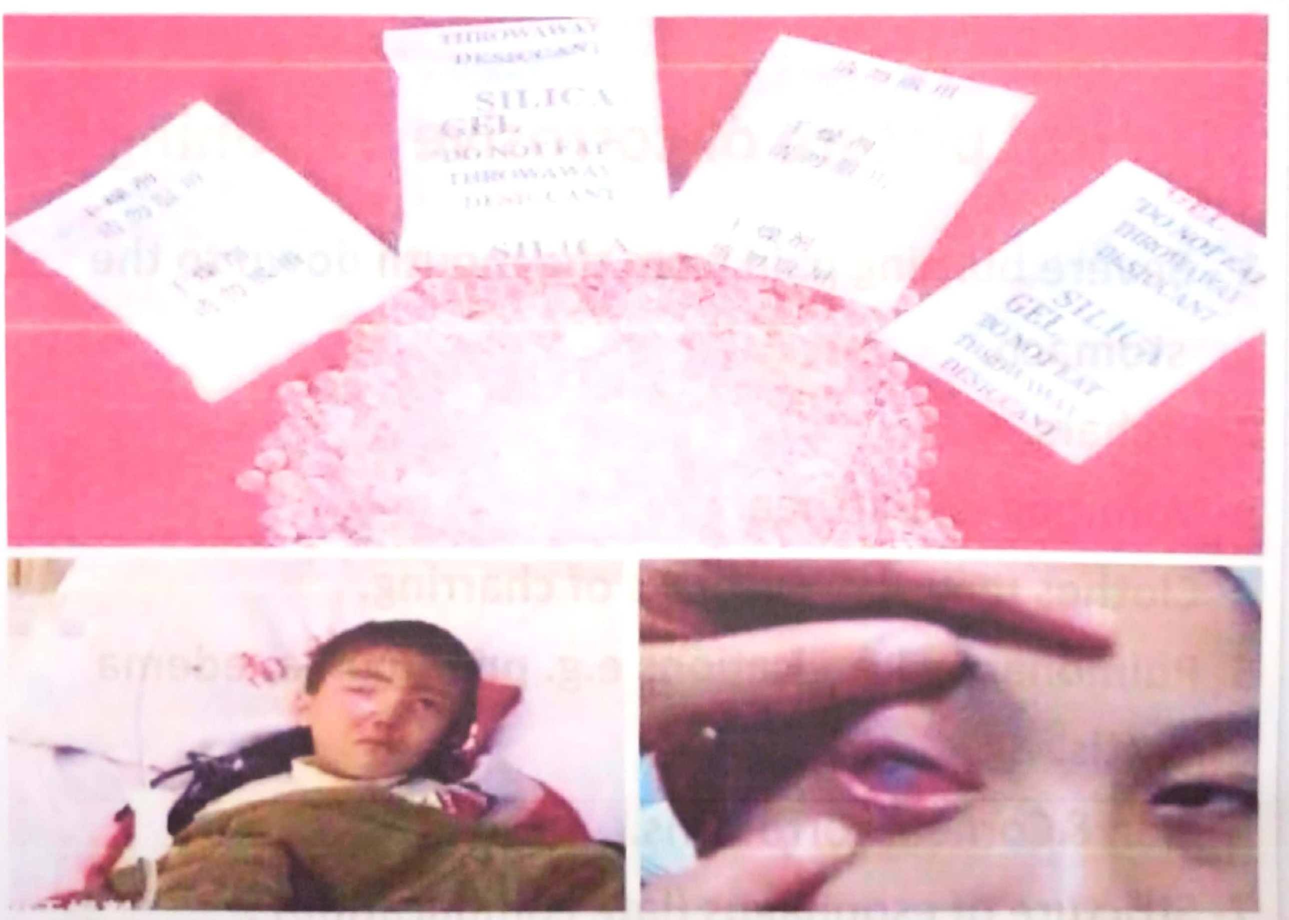
7- Stricture of esophagus (late complication) .

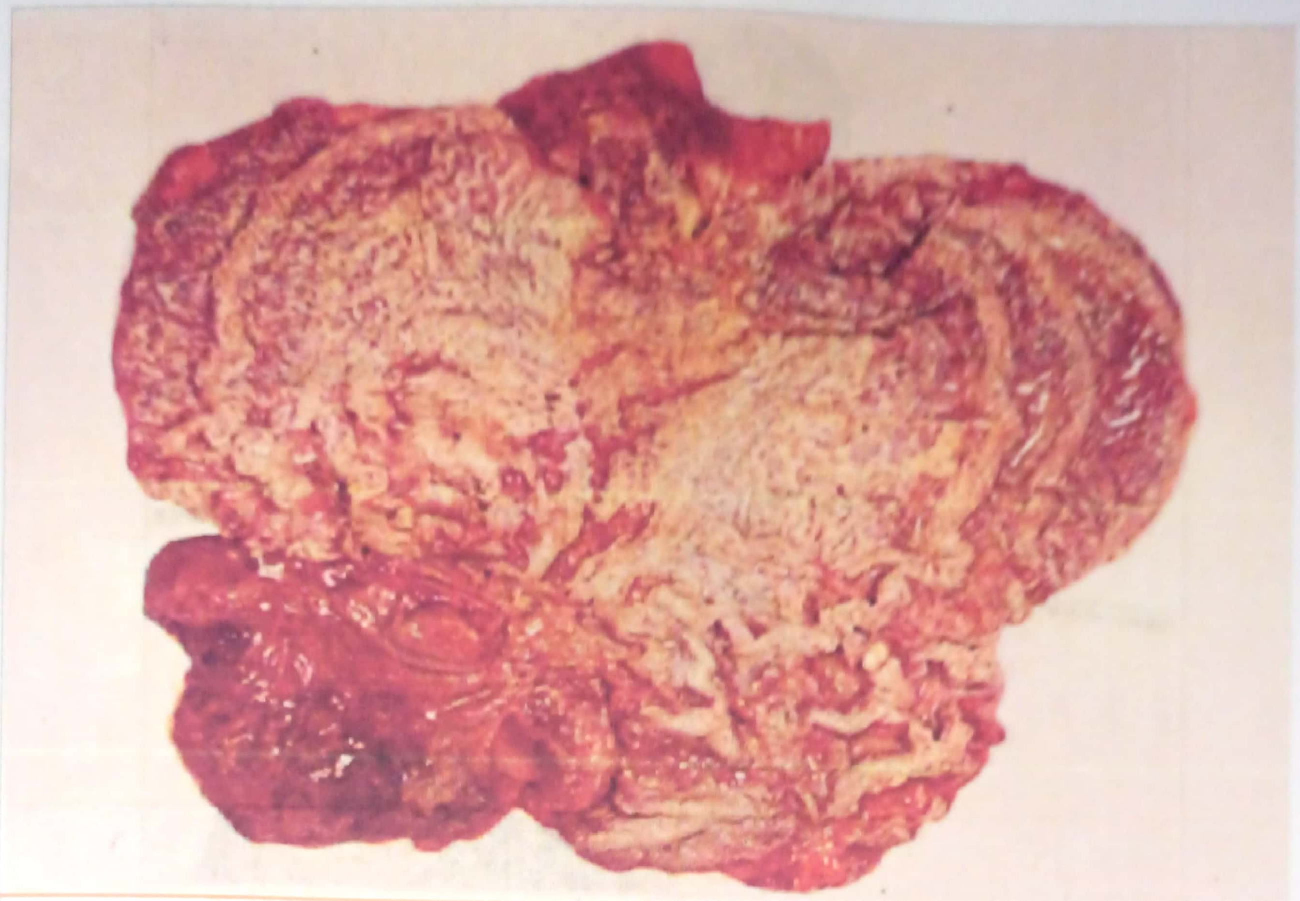


Burns of lips a case of hydrochloric acid poisoning

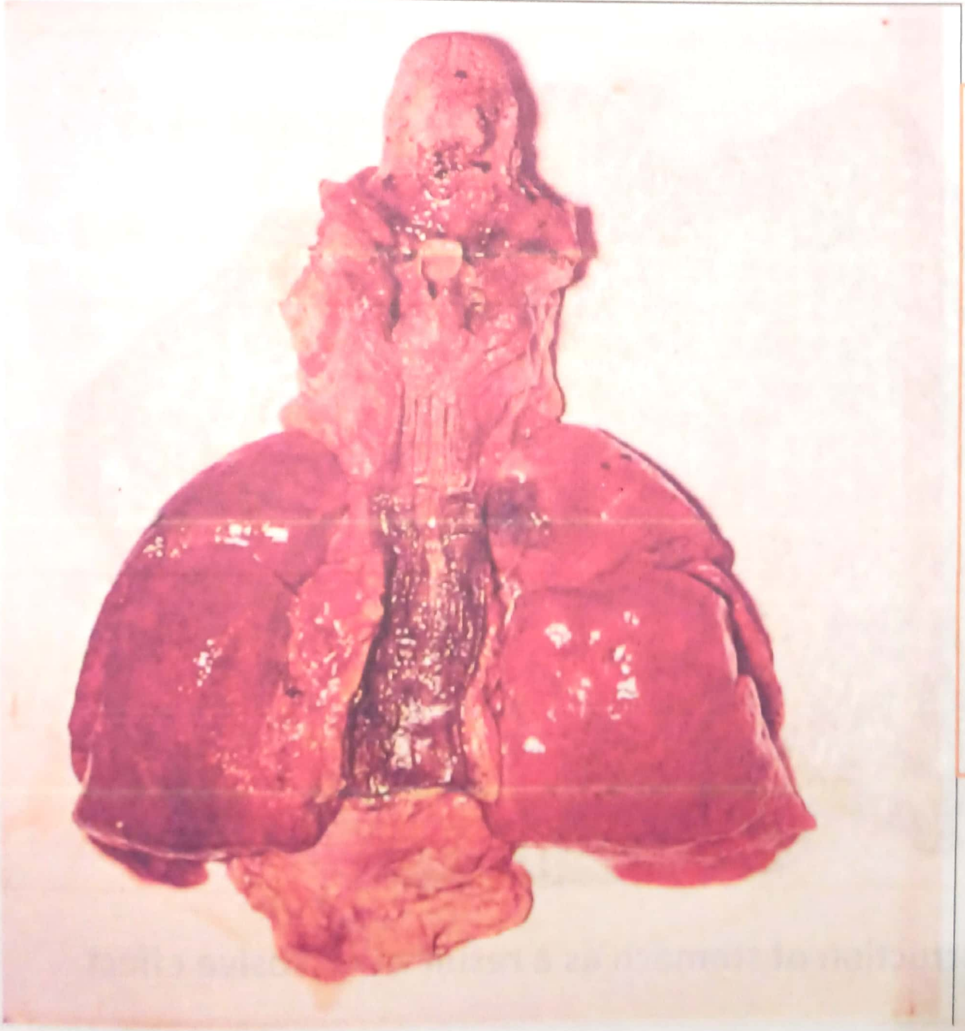


Homicide by acid caused severe burns

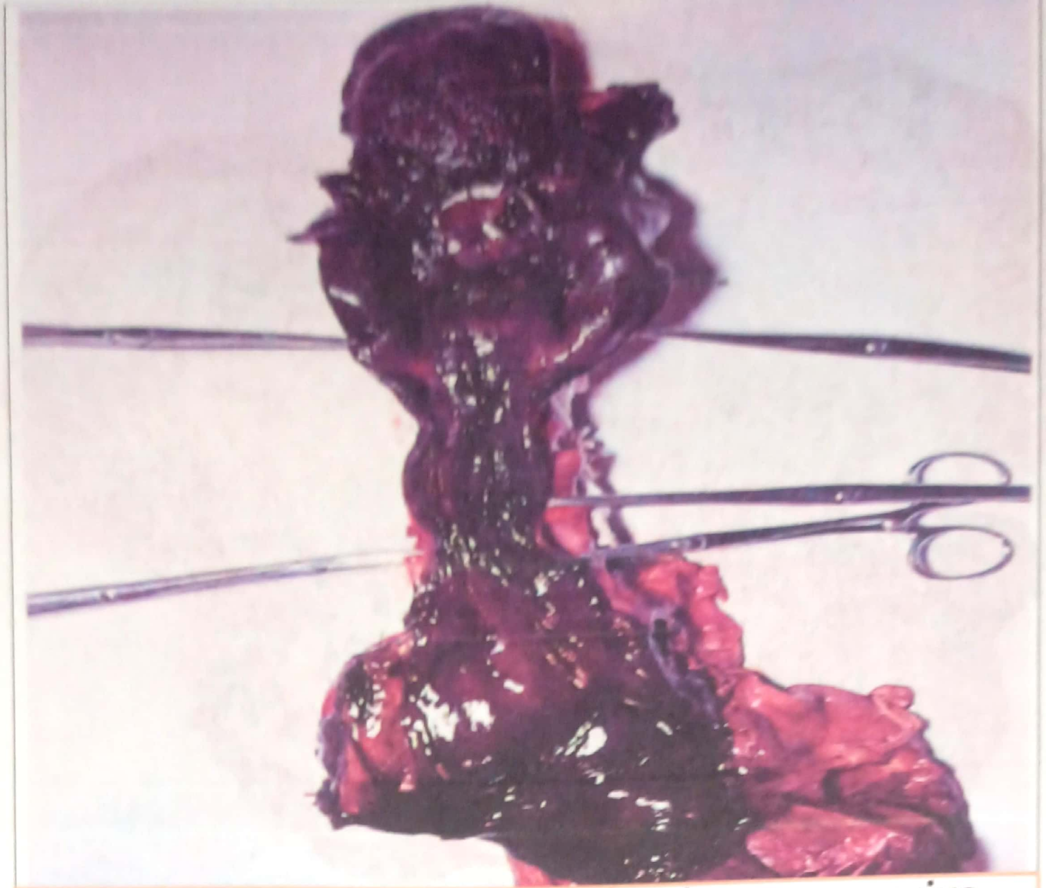

THROWAWAY
DESICCANT
SILICA
GEL
DO NOT EAT
THROWAWAY
DESICCANT



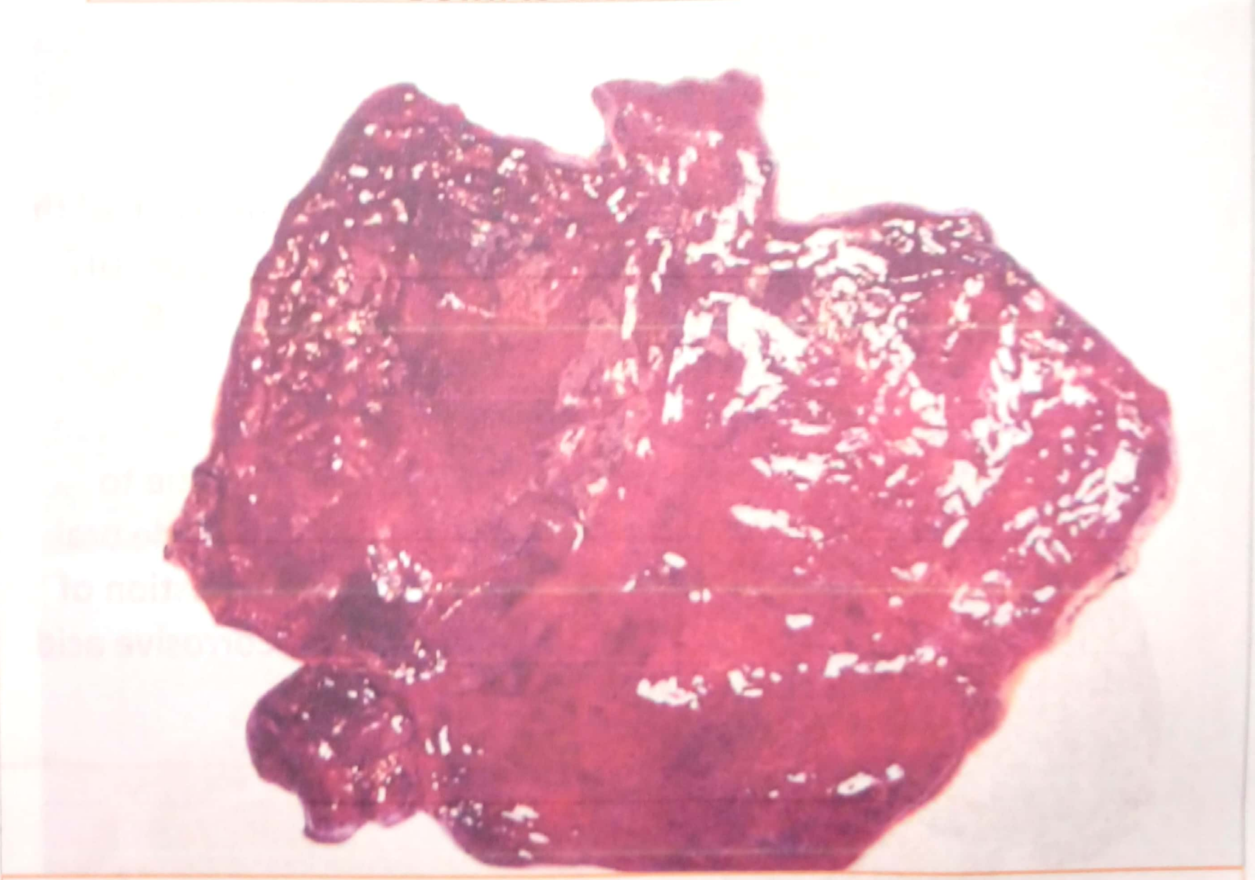
Dettol effect on gastric mucosa: thickening of mucosa with shallow ulcers (superficial)



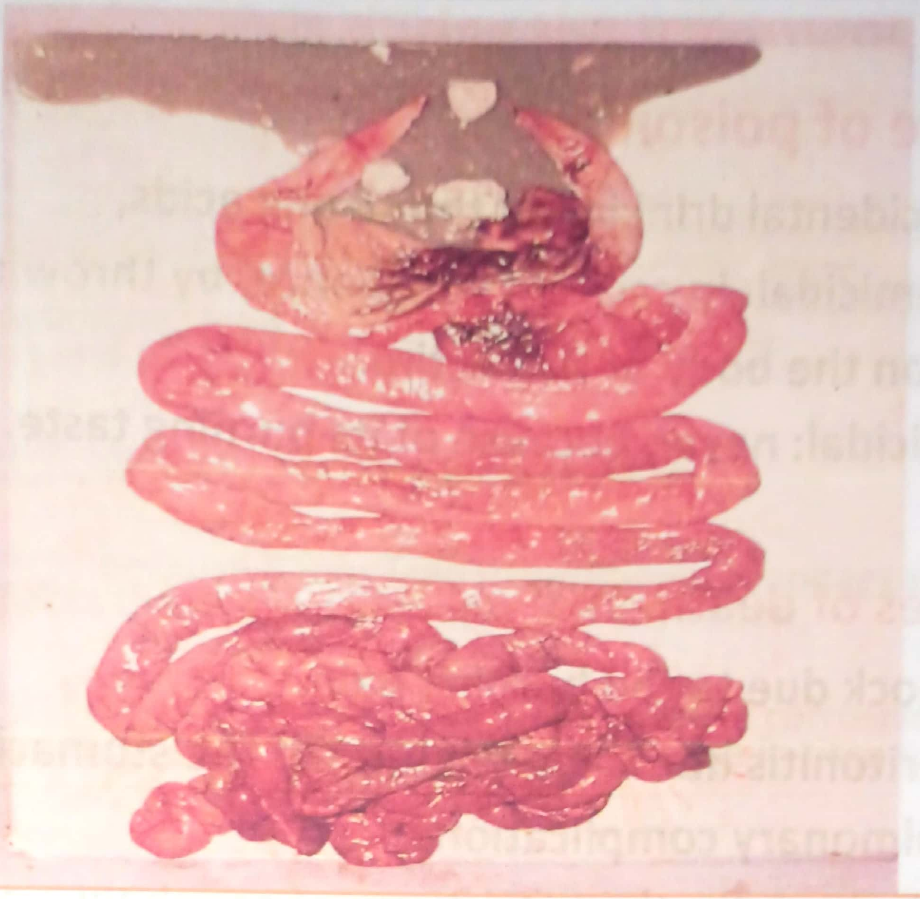
Corrosion of the upper GIT
&
Edematous congested lungs
due to
Suicide oral ingestion of Corrosive acid



Suicide by drinking ammonia: showing sever erosive effects from tongue
Down to the stomach



Mucosal destruction of stomach as a result of corrosive effect



Insecticides effect on GIT

Treatment of corrosive poisoning

1. Pain killers (Pethidine, morphine, tramadol).
2. Dilution (using water) for patients able to swallow.
3. Demulcents (using milk, egg white, oils)
4. Rehydration (IVF).
5. Cortisone (dexamethasone) to reduce edema and inflammation.
6. Wash skin and cornea without rubbing.

Mode of poisoning:

1- Accidental drinking of the strong acids.

2- Homicidal: in cases of vengeance, by throwing it on the body of the victim.

3- Suicidal: never, because of its burning taste.

Causes of death:

1- Shock due to the burning pain.

2- Peritonitis due to perforation of the stomach.

3- Pulmonary complications.

4- Late complication: strictures of esophagus and stomach.

Complications of corrosive toxicity:

1- Burns of skin

2- And/or eye destruction and loss of vision (permanent infirmity).

3- Scars of eye or mouth which remain open and exposed to inflammations or dribbling of saliva.

4- Scar in neck, twisted to one or other side.

5- Scar in limbs causing limitation of joint movements.

6- Keloid may have malignancy transformation.

Contraindications during the treatment of corrosive agents:

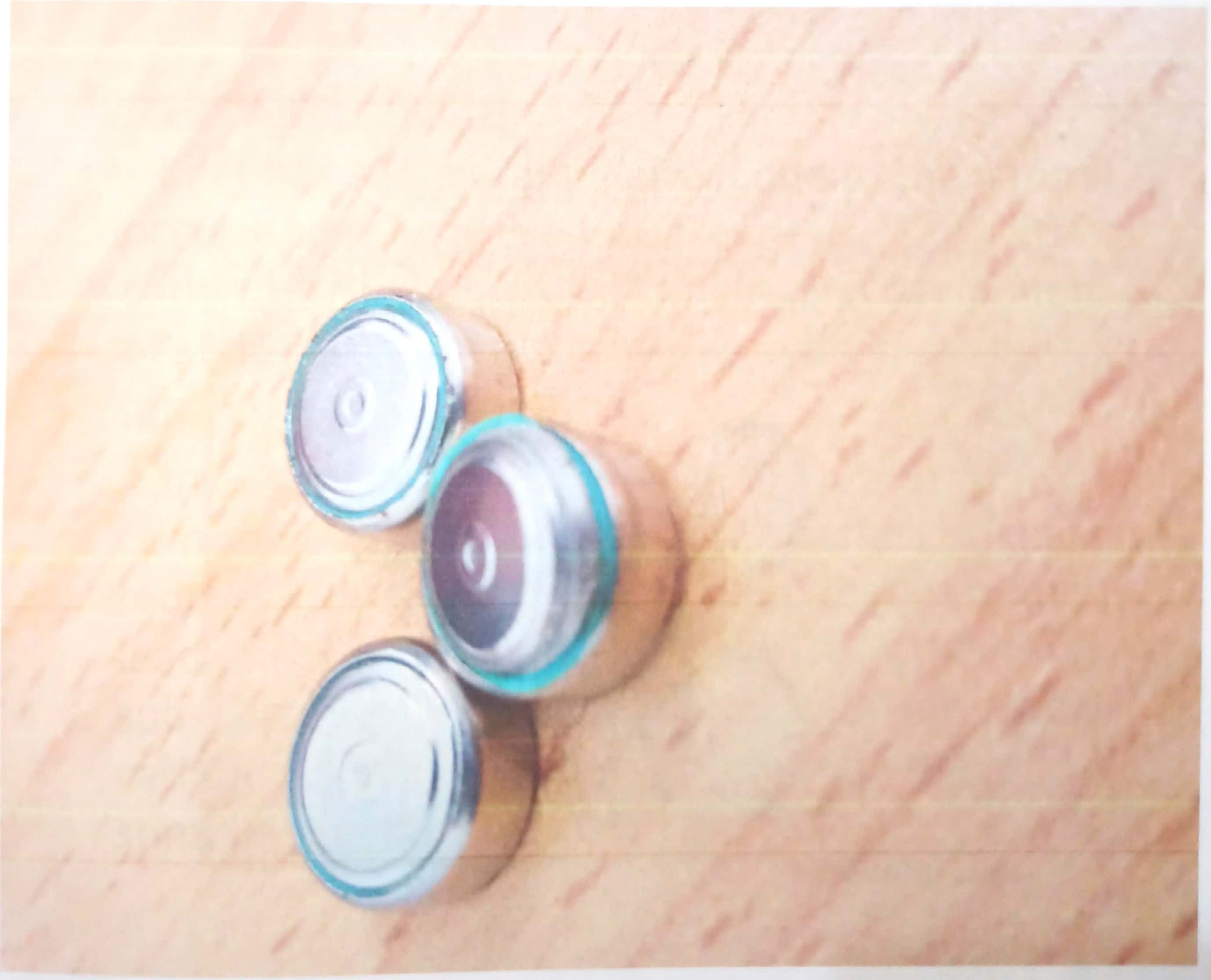
1-Neutralization: using weak alkalis in acid corrosive and weak acids in alkali corrosives are now obsolete. They cause exothermic reaction causing more tissue destruction beside carbonates lead to CO_2 production causing distension and perforation.

2-Gastric lavage: may cause perforation.

3-Emesis: because it may lead to additional esophageal injury and increases the risk of perforation and aspiration.

4-Cathartics: they enhance the movement of corrosive substances through G.I.T which leads to increase in tissue damage.

5-Charcoal: It poorly adsorbs corrosives, blocks the visual field of endoscopy and may lead to chemical mediastinitis if perforation occurs.



KHAT
(Catha)

Catha: is called as well Khat, Arabian tea, African, Miraa, or Abyssinian.

Source:

The fresh or dried leaves and stems of catha edulis, most of khat components are alkaloids.

Cultivation:

The plant is widely cultivated in the southern part of the Arabian Peninsula (Yemen) and certain parts of Africa (Kenya, Ethiopia, Somalia), it grows throughout the year at different attitudes, from coastal regions to 5000-6000 feet above sea level, and in variety of climatic conditions and soils. The tree ranges from less than a meter to more than 6 meters in length for a fully grown tree.

Toxicological effects:

- Khat has a stimulating effect due to the natural occurrence of cathine (norpseudo-ephedrine), cathine is the only well characterized component of khat. It is amphetamine like substance (phenylalkyloamino-type).
- A fresh and air dried khat samples were compared, it was noticed that the effect of khat is reduced greatly in the second and third day, this lead to the conclusion that there is another substance stronger than cathine, withch is a new cathine substance (cathinone) causing the stimulating effect of khat.

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- **According to W.H.O isolation and characterization of 27 components were reported as follows:**
- 6 are norpseudoephedrine compounds.
- 15 are high molecular weight polyester type alkaloids called cathedulins which are weak bases in addition to some acids(acetic, benzoic, gallic, nicotinic acid ascorbic acid and oxalic acid.
- 6 are neutral substances.

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- Cathinone is highly unstable and easily decomposed to weaker components, particularly when exposed to air, that's why khat must be always kept wet after being cut from its tree.
- **It was found that cathinone concentration is responsible for the main effect of khat,** so khat types are known by having different effects depending on their cathinone content.

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Types of Khat:

Depends on the concentration of Cathinone:

١. الشامي اقلها احتواء للكاثينون واقل اعراضا جانبية.

٢. الأرحبي

٣. الحداء

٤. المسوري

٥. ....

٦. ....

٥١.السوطي

٥٢. الهراري اكثرها احتوا على الكاثينون واكثرها اعراض جانبية.

Clinical picture:

Stimulation followed by depression, the peak plasma level occurs after 1.2 hour from the start of chewing the plant.

Stimulation:

Occurs during the chewing period. The khat consumer is very social during this stage, and very easy to deal with.

- Mild euphoria.
- Hyperactivity.
- Mood elevation and alertness.
- Tachycardia.
- Mild increase in blood pressure.
- Dilatation of pupils.
- Mild increase in temperature.

Depression:

Occurs after the chewing period ends.

- Depression: It is difficult to deal with the khat consumer during this stage.
- Insomnia.

- On long term use the person experiences spontaneous seminal secretions, and impotence of some cases were reported.
- Toxicity may happen by chewing a freshly sprayed khat by insecticides.

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Dependence:

- Proved not to have an addiction effect, where the khat consumer have no withdrawal signs, when stops chewing khat during his travel for long time outside the sources of khat regions.
- Habituation and psychic dependence must be only applicable to khat.

Excretion:

- Cathine is excreted **unchanged in the urine 30-50 minutes** after ingestion, **40% is recovered in the urine within the first 6 hours,** and **trace amounts are detected 24 hours later.**

Treatment:

Khat has no immediate poisonous effects other than withdrawal symptoms few hours after consumption, Hararri type from Etheopia may sometimes have sever stimulating and depressing effects for those not accustemed to use it .

Hashish(Cannabis, Marjuana)

Source:

Plants:

- Cannabis Indica,
- Canabis Sativum,
- Marjuana.

Active substance:

Tetrahydrocannabinol present mainly in flowering tops (85%) & in buds and leaves(15% called **Bhango**).

حشيش

Consumption:

1. Drunk in coffee and tea
2. Smoked
3. Eaten as Manzoal (Hashish, datura seeds,sugar, honey, nutmeg & spices.
4. Cannabis tea (IV cannabis)

Symptoms and signs:

1. euphoria, sense of well being
2. Voracious appetite
3. Loss of judgments to time and space
4. **Sever abdominal pain and CNS in cannabis tea.**

Treatment: Symptomatic

الأثر الاجتماعي للمخدرات

- مشكلة المخدرات من اخطر المشاكل الصحية والاجتماعية والنفسية التي تواجه العالم أجمع وطبقا لتقديرات المؤسسات الصحية العالمية يوجد حوالي ٨٠٠ مليون من البشر يتعاطون المخدرات أو يدمنونها كحد ادنى وقابل للزيادة المخيفة وبشكل سريع.
- ولو استخدمت الإنسانية ٢٠% من الأموال المتداولة بتجارة المخدرات الدولية وغيرها من السموم الاجتماعية لاختفت الأمية من العالم ؟؟
- أما ٤٠% من تلك الأموال فهي كفيلة بمكافحة الجوع نتيجة (التصحر) في كل أرجاء العالم و ٦٠% من تلك الأموال تقضي على الفقر في ٢٧ دولة هي الأكثر فقرا من بين دول العالم .
- و لقد تضافرت عديد من العوامل السياسية ، الاقتصادية والاجتماعية لتجعل من المخدرات خطرا يهدد العالم أو كما جاء في بيان لجنة الخبراء بالأمم المتحدة " إن وضع المخدرات بأنواعها في العالم قد تفاقم

- بشكل مزعج وأن المروجين قد تحالفوا مع جماعات إرهابية دولية لترويج المخدرات كما أن شبابنا لا شك مستهدفون من قوى الشر ، بيد أن لدينا القدرة والمرجع في ديننا الحنيف ولنذكر جميعا قوله تعالى **[ولا تقتلوا أنفسكم إن الله كان بكم رحيما] ومن قوله ﴿ولا تلقوا بايديكم الى التهلكة]**
- فكثير من الناس يعتقدون ان التعود هو نوع من أنواع الادمان وهذا اعتقاد خاطىء ولابد من تعريف الادمان:
- **تعريف الادمان :** و الإدمان على مخدر ما ، يعني تكون رغبة قوية وملحة تدفع المدمن إلى الحصول على المخدر وباي وسيلة وزيادة جرعته من آن لآخر ، مع صعوبة أو استحالة الإقلاع عنه سواء للاعتماد (الإدمان) النفسي أو لتعود أنسجة الجسم عضويا (Drug Dependency) وعادة ما يعاني المدمن من قوة دافعة قهرية داخلية للتعاطي بسبب ذلك الاعتماد النفسي أو العضوي .

IRRITANT POISONS

Irritant poisons are classified into:

1. Metallic poisons: e.g., arsenic, mercury, , lead phosphorus, iron, antimony …..etc
2. Non metallic poisons: e.g., organophosphorus compounds.

Common characters of Metallic poisons:

1. **Have double action: local and systemic.**
2. **Have acute & chronic effect.**
3. **Toxic action appears after a latent period.**
4. They are **excreted mainly in the urine.**
5. They are **cumulative.** The excretion of a single dose of an irritant poison may continue for few weeks.
6. Many of them are **re-excreted in GIT**, as mercury is re-excreted in caecum, arsenic is re-excreted in the descending colon.
7. They are **not affected by putrefaction.**
8. Have acute and chronic picture of poisoning.
9. **Stomach tube can be used** in the treatment of acute poisoning.

ARSENIC POISONING

Arsenic compounds:

1. **White arsenic:** (arsenic trioxide): White crystalline, soluble and toxic.
2. **Yellow arsenic:** (arsenic sulfide): The pure form is insoluble and non toxic.
3. **Organic Arsenicals**: were used for treatment of syphilis, currently the arsenic –containing drugs Mel B is a treatment option for African trypanosomiasis.

4. **Inorganic**:
   - Arsenites: e.g., Paris green (Cu aceto-arsenite), present in Fowler's solutions.
   - Arsenates: sodium arsenate is used as a red dye, Lead arsenate is used as an insecticides.
5. **Arsine gas:** This is a very toxic gas, a colorless gas with garlic odor.

   Most of arsenic in the surface and ground water is mostly a mixture of arsenite and arsenate.

   Arsenic is also used in glass and ceramics as metal alloy and in semiconductors and other electronic devices.

NON METALLIC IRRITANT POISONS
(Pesticides)

Any substance or mixture of substances having the capability of destroying the life of any pest such as insects, rodents, fungus or weeds.

Classification:

Pesticides are classified into:

1. **Insecticides: kill insects.**
    - Organophosphorus.
    - Organochlorine.
    - Carbamate.
    - Botanical, e.g. Nicotine, pyrethrum.

2. **Herbicides: control harmful plants.**

3. **Rodenticides**: **kill rats and mice.**

4. **Fungicides: control fungi.**

5. **Biocides: to control mosses.**

6. **Moth repellent: for moth.**

1. INSECTICIDES:

Organophosphorus compounds

These compounds are mostly used as insecticides against agricultural insects. They are used in the form of spray or dusting powder.

Chemical names of orgnophosphorus compounds:

- Parathion (a famous name in Egypt is police el-nagda).
- Malathion.
- Diethyl paranitrophey thio phosphate.
- D.F.P : Di isopropy Fluro phosphate.
- T.E.P.P : Tetra Ethyl pryo phosphate (most toxic).

- H.E.T.P : Hexa Ethyl Tetra phosphate.
- O.M.P.A : Octa Methyl pyrophosphon Amide.
- Azinphos-methyl.
- Bromophos-ethyl
- Carbophenothion
- Chlorfenvinphos
- Cythioate
- Demeton-S-methyl
- Diazinon
- Dichlorvos
- Dimethoate
- Fenitrothion
- Fenthion
- Formothion
- Heptenophos
- Jodfenphos (iodofenphos)
- Mevinphos
- Phorate
- Phosmet
- Phoxim
- Pirimiphos methyl

PARATHION

Absorption:

Occurs through the intact skin, mucous membranes or lungs.

Mode of poisoning:

- Accidental: most common for the workers during spraying or during manufacturing.
- Suicidal also common.
- Homicidal: can be mixed with flour.

70 – 80% of the poisoning is accidental and is common in agricultural and industrial workers.

Action:

It crosses the blood brain barrier. Organophosphorous compounds are potent inhibitors of acetylcholinesterase and pseudocholinesterase. The inhibition of these enzymes is due to irreversible binding of phosphate radicals of organophosphates to active sites of enzymes.

In case of carbamates this binding is irreversible. The toxicological effects are due to paralysis of neurotransimission at cholinergic synapses. The cholinergic synapses are present in CNS, somatic nerves, autonomic ganglion, parasympathetic nerve endings and some sympathetic nerve endings like in sweat glands. The signs and symptoms due to muscarinic and nicotinic effects appear within a few minutes to a few hours (average 6-8 hours) to a maximum period of 24 hours

1. **Muscarinic action:** due to their anticholinestrase effect on the cholinestrase present in the R.B.Cs and in the nervous tissue, leading to accumulation of acetylcholine at the cholinergic nerve endings causing: Salivation, sweating, pallor, constriction of pupil, slow pulse, bronchospasm, increase bronchial secretions, vomiting and abdominal colic. (Pseudocholinesterase is found in serum and liver, the toxicologic effect of organophosphates are almost due to the inhibition of acetylocholinesterase in the nervous system).

   This muscarinic effect can be antagonized by atropine.

2. **Nicotinic action:** due to accumulation of acetylcholine at the myoneural junction, leads to muscular fasciculations in the muscles of the face and become generalized convulsions, leading to muscular fatigue which leads to respiratory failure.

This effect is not antagonized by atropine.

C.N.S effects: stimulation followed by depression.

Fatal dose: 20-40 mg of parathion.

Fatal period: 2 hours, it may be longer.

Causes of death:

Respiratory failure and asphyxia due to:

1. Paralysis of respiratory center (central effect).
2. Paralysis of respiratory muscles (nicotinic effect).
3. Bronchospasm, increase bronchial secretion and pulmonary edema (muscarinic effect).

Treatment:

A. Prophylactic for the workers in contact with insecticides:

1. Wearing protective clothes, long boots and gloves.
2. Spraying must be through a long nozzle.
3. Workers should not eat, drink or smoke during work.
4. Washing must be done after finishing.

B. Curative:

1.Antidotal therapy: (Atropine and oximes).

a. Atropine: life saving physiological antidote.

Dose: 2- 5 mg atropine sulphate I.V slowly followed by 2-5 mg I.M every 5-10 minutes in adults, and in children 0.05 mg /Kg. Atropine is given till full atropinisation, this is known by:

- Pulse is getting back to normal, dryness of secretions and disappearance of pulmonary edema, pupillary dilatation (pupils must be reactive) and flushing of the skin.

Advantages of atropine:

It antagonizes the muscarinic effect, it is respiratory stimulant and it dries up the secretions.

b. Oximes (cholinesterase enzymes reactivators): given for reactivation of the inhibited cholinestrase enzyme, must be given in the first 24 to 48 hours.

Given to all patients with severe toxicity (muscle weakness and respiratory depression). It rapidly ameliorates muscle weakness, fasiculations and coma.

Dose: 1-2 gms in adults or 25-50 mg/kg in children over 5-10 minutes IV. The dose may be repeated after 1 hour.

Contraindicated with opiates, phenothiazines and parasympathominetic durgs.

They include:

- P.A.M (pyridine aldoxime methiodide) or pralidoxime chloride (Protopam), 1g I.V slowly.
- D.A.M (Di-acetyl Monoxime): 1gm I.V slowly.
- Obidoxime (Toxogonin): 0.5-1gm I.V.

2. When ingested wash the stomach with 2% potassium permanganate.
3. Treatment of convulsions: using diazepam or thiopental I.V.
4. The skin must be washed if contaminated.

Laboratory investigations:

Although physicians recommend cholinesterase estimation before starting treatment. DO NOT WAIT for cholinesterase estimation before starting treatment.

1. Latent poisoning (Serum cholinesterase activity 50-90% of normal value) no treatment is needed.
2. Mild (20-50% of normal value), treatment is needed.
3. Moderate (10—20% of normal value).
4. Severe (serum cholinesterase less than 10% of normal).

ORGANOCHLORINE INSECTICIDES

Examples:

D.D.T(Dichloro-Diphenyl-Trichloroethane), Endrin, Aldrin, Chlordane, Dieldrin, Endosulfan, Lindane (also known as gamma benzene hexachloride or gamma-HCH), Toxaphene and Gammaxane.

They are fat soluble stable compounds (not water soluble),thus diffuses in the fat tissues affecting spinal cord and brain cortex.

Uses:

Used at home to control house flies (malaria mosquitoes bed bugs, fleas and head lice). Lindane shampoo can cause poisoning in young children if too much is used or if it is used too often. Rarely used as field insecticide.

Fatal dose: 10-20 gm.

Clinical feature:

- C.N.S: stimulation followed by depression:
  Restlessness, tremors, tonic convulsions followed by depression leading to paralysis of muscles then respiratory failure.
- GIT: Severe vomiting, colic, diarrhea.

Treatment: No specific antidote.

1.Gastric lavage for cases of ingestion.
2.Treatment of convulsions.
3.Symptomatic treatment.

CARBAMATE COMPOUNDS

Examples: Carbaryl (sevin), propoxur (Baygon), methomyl (Lannate) aldicarb, bendiocarb, carbofuran, methiocarb, primicarb and Dixan.

Action and clinical picture:

Same action and clinical picture as organophosphorus insecticides, but they have shorter duration of action and they are less toxic.

Treatment:

Is the same as organophosphorous, Atropine is the only antidote. oximes are contra -indicated because spontaneous reactivation of cholinesterase is more rapid after inhibition by carbamates..

BOTANICAL INSECTICIDES

Examples: Nicotine, rotenone, pyrethrum. They are derived from natural products, they are less toxic to mammals than synthetic insecticides.

Pyrethrum is the same as RAID and used as a household insecticide.

Clinical picture: Severe poisoning is rare, inhaled pyrethrum may cause:

- Nausea vomiting, asthma and rhinitis in allergic persons.
- Severe poisoning may cause convulsions, muscle paralysis and death.

Treatment: Is symptomatic.

BIPYRIDYL HERBICIDES

Examples: Paraquat and Diquat (Gramozone).They destroy weeds and plant tissues if they come in contact with plants, they are rapidly inactivated if they come in contact with soil. They are sprayed for one day to destroy broad leaf weeds, and planting new seeds on the next day.

Action:

They are responsible for inhibition of superoxide dismutase enzyme in the lungs.

Clinical picture:

It has 3 stages:

Stage I: (1-5 days): Corrosive effect, nausea, vomiting diarrhea and haemoptysis.

Stage II: (2-8 days): Fever, jaundice, increase blood urea and creatinine, arrhythmias and heart failure.

Stage III: (3-14 days): Signs of pulmonary fibrosis, pleural effusion and respiratory failure.

Treatment:

1. Gastric lavage with 30% Fuller's earth, 6% to 7.5% suspension of bentonite, or activated charcoal. Lavage should be repeated every 2-4 hours followed by cathartic as sodium sulphate.
2. Foreced diuresis, hemoperfusion.
3. Reducing pulmonary damage by glucocoritcoids , Vit C and E.

RODENTICIDES

Example: Warfarin , zinc phosphide and aluminium phosphide.

Warfarin:

Action:

It is an anticoagulant, it inhibits synthesis of vit. K dependent clotting factors in the plasma and liver. Thus prothrombin synthesis is reduced. Multiple doses are required to inhibit synthesis, until prtohrombin level is sufficiently depleted, resulting in hemorrhage in the whole body leading to death.

Clinical picture:

Symptoms begin few days or weeks of repeated ingestion

1. Epistaxis.
2. Bleeding gums.
3. Pallor.
4. Petechial rash.
5. Haematoma around joints and on the buttocks.
6. Haematurea.
7. Melina.
8. Cerebral hemorrhage.
9. Death from hemorrhagic shock.

Treatment:

1. Vit. K (phytondione).
2. Fresh blood or plasma transfusion in sever cases.

Zinc phosphide and aluminium phosphide:
Zinc and Aluminium phosphide are used to preserve grain, especially wheat, and to kill rats. Grain preservative is usually sold as tablets, and rat killer is sold as pellets or bait.
When damp, phosphides release poisonous phosphine gas. When swallowed, poisonous effects are due to phosphine released in the gut.

Mostly accidental, and rarely homicidal.
It is luminous in the dark.

Fatal Dose: 0.2 gm of yellow phosphorous.**Action:**
Local on GIT: irritation.
Systemic:
-Fatty degeneration of the liver with hemorrhages and jaundice.
-Pulmonary edema.
-cardiac arrhythmias.

Cause of death:
After 24 hours: collapse from GIT irritation.
After 10 days: Cholaemia from toxic effect on the liver.

Treatment:
1.Gastric wash with potassium permanganate (to oxidize phosphorous into
non poisonous phosphate, oily laxatives and fats are contra-indicated.
2.Treatment of liver failure.
3.Treatment of pulmonary edema.
4.Treatment of acidosis, anoxia and dehydration.

No specific antidote is available. Since phosphine, is excreted through breath and urine, adequate hydration and renal perfusion by low dose dopamine 4-6 mg/kg/ min is to be used. Magnesium sulfate can be used as an antioxidant and is also useful as an anti-arrhythmic and anti - hypoxic agent.

A dose of 1 gm of magnesium sulfate IV, stat is given followed by 1 gm for next 2 hours and finally 1-1.5 after 4-6 hours for 3-5 days or final outcome of these patients.

P.M.P:

Inflamed gastric mucosa, garlic smell and enlarged fatty liver with hemorrhages.

ATROPINE

Source:

Atropine, hyoscyamine and hyoscine are present in all parts of the plants:

1. Atropa beladona.
2. Hyoscymus Nigers.
3. Datura stramonium and fastiosa.

Action:

Atropine and hyoscyamine have the same pharmacological action, but atropine is much more powerful.

1. Central action on the C.N.S which lead to stimulation followed by depression.
2. Peripheral action: Blocking of the muscarinic action of acetylcholine at the cholinergic nerve endings, including the sympathetic nerve endings (cholinergic) which supply secretary fiber of the sweat glands.

Mode of poisoning:

1. Usually accidental:
    - Children eating the berries of atropa belladonna, or seeds of datura.
    - Taking drugs containing atropine.
2. The crushed seeds may e mixed with dates and used to facilitate robbery or rape.

Clinical features:

I. The peripheral action produces:

1. A diminish of all body secretions.
    - Dry mouth, hoarseness of voice and dysphagia.
    - Dry skin due to absence of sweating.
2. Slight rise n temperature and flushed skin due to peripheral vasodilatation, and no loss of heat due to loss of sweating.
3. Pupils are dilated and fixed leading to loss of accommodation to near objects.
4. Rapid weak pulse and shallow respiration.

II. Central action:

In large doses atropine causes stimulation:

- Excitation.

- **Purposeless movement.**
- **Restlessness.**
- **Convulsion.**

In small doses causes depression:

- **Decrease cerebral cortex → sleep, stupor and coma.**
- **Depression of medulla → decrease pulse and respiration leading to death.**

Fatal dose:

100-150 mgs.

100 datura seeds.

Treatment:

1. **Stomach wash with charcoal suspension or potassium permanganate 1 in 5000.**
2. **Sedation to control cerebral excitation: diazepam 5-10 mg I.V slow or short acting barbiturates. Morphine and other narcotics are contraindicated because they aggravate respiratory depression.**
3. **Specific antidotes:**
   - **Physostigmine (Eserine): 1-5mg I.V → reverses both central and peripheral effects of atropine.**
   - **Pilocarpine: Reverses only the peripheral effect.**

VOLATILE POISONS

Ethyl alcohol (Ethanol)

It is colorless liquid with characteristic odor. It is obtained from fermentation of food e.g. barley, bread or fruits. The percentage of alcohol in beverages varies according to the type of alcoholic beverages.

Ethanol is the most common ingested toxin throughout the world, thousands of deaths occurs due to overdose, suicide and accidental intake of alcohol.

Alcoholic beverage:

The alcoholic content of different beverages are:

- Beer: 2-8%.
- Light wines: 5-10%.
- Heavy wines: 10-20%.
- Brandy, Rhum (rum), Vodka and Wisky: 40-50%.

Absorption and elimination:

- Ethyl alcohol can be absorbed by the mucus membrane of the stomach and the upper part of the small intestine.

- Absorption occurs more rapidly when its taken on an empty stomach. Its delayed by the presence of food, especially fatty food.
- After absorption: It reaches its maximal concentration in the blood after 0.5-1 hour.
- Abut 90% of the amount absorbed is oxidized in the body into acetaldehyde and then into carbon dioxide + water + energy. The remaining 10% is excreted unchanged in the urine and breath.
- The rate of oxidation of alcohol in the body after absorption is 0.1ml/hg/body wt/hour.
- The concentration of alcohol in the blood can be indirectly estimated by measuring it's concentration in alveolar air by alcometer.

Metabolism:

Ethanol is oxidized to acetaldehyde by alcohol dehydrogenase and then metabolized into $C0_2$ and water, this is considered the main path of metabolism, microsomal ethanol oxidizing system (MEOS) plays a minor rule.

Because of mucosal and hepatic metabolism, the oral dose yields a lower blood ethanol level than in equivalent administered I.V dose.

Effects:

-Hypoglycemia mainly in children and in chronic use.
-Hypomagnesaemia.
-Alcoholic ketoacidosis.

Action:

1. Mainly on the CNS, leading to depression of the cerebral cortex and reticular activating system..
2. Gastric irritation.
3. Dilatation of skin vessels.
4. Slight depression of the liver function.
5. Arrhythmia and cardiomyopathy on chronic use.
6. Peripheral neuritis.

Fatal dose:

- 150 – 200 mls of absolute ethyl alcohol.
- Barbiturates & tranquilizers enhance (synergism). the fatal effect of alcohol.

Fatal period: 1-10 hours after intake of fatal dose.

Picture of acute alcoholism:

Toxic manifestations begin to appear when the concentration of alcohol in the blood reaches **0.15%** **(Moderate intoxication):**

a) Alcohol euphoria (sense of well being and self confidence).
b) Loss of power of concentration.
c) Change in personal behavior → A drunken person may become aggressive, or may commit sexual crimes.
d) Flushing of the skin.
e) Increase pulse and respiration.

In a concentration of **0.25%** in the blood:

f) Staggering gait (Loss of muscular coordination).
g) Slurred speech.
h) Blurred vision, diplopia and nystagmus.
i) Tremors of hands and lips.
j) Hiccough.

k) Vomiting due to central effect of acetaldehyde.

l) Gastric irritation by concentrated alcohol.

m) False sensation of heat, due to dilatation of skin arterioles.

When blood alcohol reaches **above 0.3%** **(Severe intoxication):**

a. Skin becomes pale with profuse sweat.

b. Subnormal temperature.

c. Rapid weak pulse.

d. Constricted pupils: Dilate on pinching the skin of face (McEwen's sign).

More than 0.35% blood alcohol = dead drunk.

More than 0.5% → Death from central respiratory failure.

Treatment:

a. Aspiration of stomach contents, and wash by sodium bicarbonate, then leave strong coffee in the stomach (stimulates respiratory center).

b. Clear air passages.

c. O_2 inhalation.
d. Respiratory stimulants.
e. I.V **hypertonic glucose to** decrease brain edema.

METHYL ALCOHOL (Methanol)

Methyl alcohol is widely used in industry and laboratories and hospitals as a solvent. Many cases of poisoning occurs due to adulteration of ethyl alcohol by adding methyl alcohol, or methyl alcohol is taken as a substitute for ethyl alcohol.

Metabolism:

Methyl alcohol is metabolized mainly in the liver $\xrightarrow{\text{by dehaydrogenases}}$ to formaldehyde and formic acid, both are more toxic than methanol, leading to blindness and acidosis.

Fatal dose;

60-150 mls → 15 mls is enough to cause visual effect.

Action:

CNS depression, cyanosis, metabolic acidosis, retinal edema, optic neuritis, optic atrophy and blindness.

Fatal period: Variable

Clinical picture:

There is a latent period of about 12 hours between ingestion and onset of symptoms, this period might extend to 72 hours and is due to the slow rate of production of formaldehyde and formic acid.

1. Headache.
2. Visual symptoms: Blurred vision and flushing lights (visual acuity may diminish to perception of light or blindness)
3. Violent attacks of abdominal pain due to pancreatitis.
4. Vomiting.
5. Drowsiness leading to coma.

Treatment:

1. Stomach wash, if more than 20 ml has been ingested.
2. I.V sodium bicarbonate to correct metabolic acidosis.
3. Antidotal therapy: as soon as methanol ingested.

a. Ethanol, because the enzyme (alcohol dehydrogenase) oxidizing methanol prefers ethanol if both alcohols are present.

- In moderate cases: oral intake (0.5mg/kg of ethyl alcohol is given followed by 0.25ml/kg 2 hourly.
- In severe cases: I.V 5-10g/hour to reach a plasma ethyl alcohol concentration of 1-2g/l.

b. Fomepizole (4-methyl pyrazole): is the preferred antidote because its ease to use (oral and I.V) can be given to patients as home therapy, it has no C.N.S depression, no hypoglycemia, no hyponatrimia and no need for frequent monitoring of drug levels. It is a potent inhibitor of alcohol dehydorgenase.

4.Cofactor therapy:

Folinic acid (leucovorin) I.V 4 hourly till methanol is undetectable and metabolic acidosis has cleared.

5.Hemodylysis in serious cases.

It should be clear that the absence of signs of toxicity does not mean the patient situation is not dangerous, but death may occur later.

EHYLENE GLYCOL

Is an antirust antifreeze agent, mainly used in car radiators, it is not absorbed through the skin or respiratory system (the boiling point is197 degrees centigrade) which is high so no vapor can be inhaled.
It causes C.N.S and C.V.S effects but the most dangerous effect is renal effect.

Antidote: Ethanol.

PETROLEUM DISTILLATES

Kerosene

Kerosene is a volatile liquid, its one of the petroleum distillates, used as a fuel and solvent.
Other petroleum distillates:
Diesel oil, Gasoline, Naphtha, Petroleum ether, Benzene, Paint Thinner. All of them have similar toxic effects and treatment.

Fatal dose:
Ingestion of 20ml may be fatal.

Fatal period:

24 hours leading to central asphyxia.
Few days leading to pneumonia.

Action:

1. Kerosene has a low surface tension, so small quantities will spread over a large surface area as the lung.
2. Kerosene is an irritant, dissolves in the body fat.
3. Depresses the cells of CNS.
4. Direct aspiration of kerosene into the lungs during ingestion or during vomiting appears to be the cause of pulmonary irritation.

Mode of poisoning: Mainly accidental especially in children.

Clinical picture of acute poisoning:

1. The principal manifestation of poisoning is pulmonary irritation.
2. Cough.
3. Nausea and vomiting.
4. Pulmonary edema.
5. Bloody sputum + pneumonia + fever +cough.

6. If large amount (more than 1ml/kg) is ingested and retained → CNS depression and irritation.

Treatment:

1. Stomach wash ONLY after the insertion of a cuffed endotracheal tube, and the induction of emesis is contra-indicated due to aspiration.
2. Care of respiration.
3. Antibiotics to prevent pneumonia.

HYDROCYANIC ACID (HCN) POISONING

"Prussic acid"

The acid is a volatile liquid. The acid itself is not a corrosive, having a bitter almond smell. It's salt (KCN) has a slight corrosive action. Cyanides are the fastest poisons ever known.

Sources of cyanide:

1. Amygdalin: It is cynogenetic glucoside present in seeds of cherry, better almonds and unripe apples and gawafa.

Amygdalin in fresh state when mixed with stomach HCL it liberates

HCN by the effect of emulsion enzyme.

2. Simple cyanide salts as sodium and potassium cyanide , if combined with HCL (gastric activity) leads to the liberation of HCN. Achlorohydra does not cause toxicity.

Mode of poisoning;

1. Suicidal: common, because its rapidly fatal. (Used by spies).
2. Accidental:
    - During fumigation of ships to kill rats.
    - In electroplating → photography and engraving.
3. Homicidal: Rare, done sometimes in USA for execution of murderers in gas chamber by HCN gas.

Fatal dose:

- 1 drop of HCN.
- 300mg of KCN.
- 1/20000 HCN fumes in the inspired air.

Fatal period: 2-10 mins.

Action:

Cyanide paralysis the cytochrome oxidase enzyme → tissue anoxia (histotoxic anoxia) → the tissue will not be able to utilize the o_2 in the blood → the venous blood retain the o_2, the same as that of arterial blood → giving the bright red color.

Clinical picture:

1. Cyanide cry (due to laryngeal spasm) → patient falls down, due to anoxia → sudden coma → convulsions with froth coming from the mouth.
2. Dilated pupils.
3. Cornea keeps it's luster even after death.
4. Weak pulse.
5. Respiratory → difficult and gasping.
6. Bright red skin due to oxy Hb in venous blood.
7. Characteristic odor of bitter almond in breath.
8. Vomiting in a case of KCN poisoning.

Treatment of cyanide poisoning:

Must be very rapid:

1. Inhalation of amyl nitrate, which is life saving.

2. I.V injection of sodium nitrate, 10mls of 3% solution. Nitrates will change oxy Hb into methaemoglobin, which unites with cyanides forming the non toxic cyanmethaemoglobin.
3. I.V sodium thiosulfate, 10mls of 10% solution, to form the non toxic thiocyanates.
4. Respiratory stimulants or artificial respiration.
5. Stomach wash, using either:
    a. An oxidizing agent e.g. hydrogen peroxide, or potassium permanganate solution. This will oxidize cyanides into the less toxic oxy cyanide.
    b. Ferric hydroxide forms the non toxic ferricyanide.
6. A new antidote hydroxycobalamine (Vit. B_{12a}), 1000 microgram I.V → It unites with cyanides → forming cyanocobalamine (Vit. B_{12}).
7. Cobalt edetate can be used as an antidote. 40ml of 1.5% solution. I.V followed by 20ml, 50% glucose.

Inhalation of O_2 in the case of cyanide poisoning is ineffective, because the toxicity is due to the inability of the tissue to utilize o_2 in the blood.

P.M.P:

1. Odor of bitter almond in stomach.
2. Bright red skin and mucus membranes.
3. Bright red hypostasis.
4. Bright red hyperaemic gastric mucosa.
5. Gastric contents appear alkaline.

CARBON MONOXIDE (CO)

Characters:

Colorless, odorless gas, inflammable and slightly lighter than air.

Sources:

1. Running motor car engine, in closed garages.
2. In kilns (house + brick kilns).
3. Incomplete consumption of organic matter e.g. during fire.
4. In coal mines.
5. Leakage from coal gas pipes. (coal gas = CO + mixture of gases).

Mode of poisoning :

1. Accidental: Most common result from exposure to it's sources.
2. Suicidal or homicidal: Rare.

Action:

CO is readily absorbed by the lungs. It combines with hemoglobin, myoglobin and cytochrome oxidase system and affects cellular respiration.

1. CO unites with Hb forming COHb = Carboxy haemoglobin (cherry red), its

affinity for Hb is 250 times greater than that for O_2 → tissue anoxia.

2. COHb interferes with the release of O_2 carried by unaltered oxyHb.

Picture of poisoning:

Depends on the concentration of CO in the respired air:

1. Exposure to 0.01% CO = 20% COHb.
   a) Fatigue.
   b) Dyspnea.
   c) Tinnitus.
2. Exposure to 0.1% CO = 50% COHb.
   a) Loss of power with relaxation of all muscles of the body, so the person can't walk out of the place.
   b) Impaired hearing.
   c) Dimness of vision.
   d) Decrease B.P, rapid weak pulse.
   e) Shallow respiration → coma → death.

Complications:

If the patient is rescued and he recovers from CO poisoning, he may develop:

1. Pneumonia. 2. Liver damage. 3. Permanent brain damage.

Treatment;

1. Prophylactic → preventing accumulation of CO.
2. Curative:
    - Remove patient immediately into fresh air.
    - Care of respiration: Its better to give O_2 under pressure (3 atmospheric pressure).
    - General warmth.
    - Complete rest, to minimize the amount of O_2 utilized.
    - After recovery the patient must be kept under observation → he may be confused, and may commit suicide or homicide.

P.M.P:

1. Pink hypostasis and pink viscera.
2. Petechial hemorrhages in the brain.
3. Pulmonary edema.
4. Signs of asphyxia.

IODINE

Iodine is usually taken accidentally due to ingestion of Tincture Iodine.

Action:

- It is a direct protein precipitant that is corrosive to mucous membrane.
- In the intestine, it is converted to the less toxic iodide and rapidly deactivated by food in GIT.
- It causes a strong reflex vomiting, which removes much of the poison.

All of these factors help to minimize toxicity.

Clinical Picture:

- GIT irritation (vomiting, burning pain).
- Salivation and enlargement of salivary gland, because it is secreted by salivary glands).
- Lacrimation and conjunctivitis.

Treatment:

1. Gastric lavage using starch, to precipitate Iodine producing blue color, washing should be continued till the blue color disappears.
2. Gastric lavage by sodium thiosulphte to reduce iodine to iodides.
3. Atropine to dry secretions.

P.M.P:

- Gastro-intestinal irritation and hemorrhage.
- Skin rashes, edema of mouth, eyes and upper respiratory passages.
- Parenchymatous degeneration of glandular organs.

SNAKES (Ophidia)

There is about 2500 species in the world, they predominate in warm climates and in bush region in the tropics.
In Asia 216 snakes are called non-poisonous, but still they have enough poison to kill small animals.

Types of snakes are:

1. **Elapid snakes:**
   Cobra, King Cobra, Common Krait, banded krait and coral snake. Their fangs are small 2-4 mms, fixed and grooved therefore they can't bite through clothes, and only sublethal doses of venom is given. The head is nearly the same width of the neck. The pupils are round.
2. **Viper snakes:**
   Rattle snake, pit Viper (bamboo) and pitless Viper:
   - Russell's Viper.
   - Saw-Scaled Viper

   Their fangs are long 12-15mms, movable, canalized like hypodermic needle, therefore these snakes can bite through clothes and give lethal dose. The head is triangular and wider than the neck and the pupils are vertical. The pit lies between the eyes and nostril helps in the detection of warm blooded prey in the dark.
3. **Sea snakes:**
   Found near sea coasts, they have small heads and rudder-like tail to help in swimming. They have small fangs situated posteriorly, although they are classified as poisonous, but most of them don't bite.

Snake venom:

The normal function of snake venom is to immobilize the prey and to assist in digestion. In fresh state it is clear transparent and in dried state it is a yellow granular mass, can retain it's activity for many years, but not toxic when ingested.
Contains several toxic principle and toxalbumens such as :

1. Fibrinolysins.
2. Neurotoxins → Elapids.
3. Haemolysins → Vipers.
4. Agglutinins.
5. Coagulase, hyaluronidase.
6. Proteolysis.

7. Cholinestrase → Elapids.
8. Thromboplastins → Vipers.
9. Cardiotoxins.

Venoms:

1. **Neurotoxic:** Elapids → Sumptoms and signs appear 2-6 hours after envenomation.

Causes *disrubtion of neuromuscular activity:*

- Paralysis of face muscles (ptosis, salivation), throat (dysphagia), decrease respiration, weakness of the legs.
- Local symptoms at the site of bite is minimal. Cobra venom causes convolutions followed by paralysis, but krait venom causes only paralysis.

2. **Cytolytic(vasoculotoxic):** Vipers → 15-30 mins. Due to proteolytic enzymes:
   - Lysis of cells.
   - Haemolysis (destruction of blood vessels endothelium and increase capillary permeability).

   Local symptoms are very marked, with an extensive painful swelling which spread from the site of bite to involve the whole limb, with spread cellulites and convulsions due to cerebral haemorrhages.

3. **Myotoxic:** Sea snakes → 3-5 hours after envenomation, produces generalized muscular pain, followed by myoglobinurea (brown urine). Respiratory failure in fatal cases.

Fatal dose:

- 15 mg of dries cobra venom (yield in one bite from 200-300mg)
- 6 mg of dried krait venom (yield in one bite 22mg)
- 40 mg of dried viper venom (yield in one bite from 150-200mg)
- 8mg of saw scaled viper (yield in one bite 25mg).

Fatal period:

Death may occur within few minutes or may be delayed for few days. Generally death from cobra venom occurs within few minutes to few hours. Death from viper venom in few days. No reported cases of death from sea snakes.

Treatment:

There is no general agreement on standard procedures of treatment. The main principles are:

1. Allaying anxiety and fright.

2. prevention of spread of venom by :
   - Immobilization.
   - Blocking only venous and lymphatic return not arterial flow. (only on limbs).
3. Use of antivenin and other antitoxic therapy.
4. General measures.

Antivenin:

Two types:

1. **Specific** → prepared by hyper-immunizing horses by a venom of a particular snake.
2. **Polyvalent** → prepared by hyper-immunizing horses against the venoms of four common poisonous snakes:
   - A. Cobra.
   - B. Common krait.
   - C. Russel's viper.
   - D. Saw-scaled viper.

Specific antivenin is valuable when the identity of snake is known (attempts to bring the snake for identification may result in further bites). The mortality of all types of snake bites is about 40% and the antivenin reduces it to less than 10%, it should be administered as the given instruction on the vial.

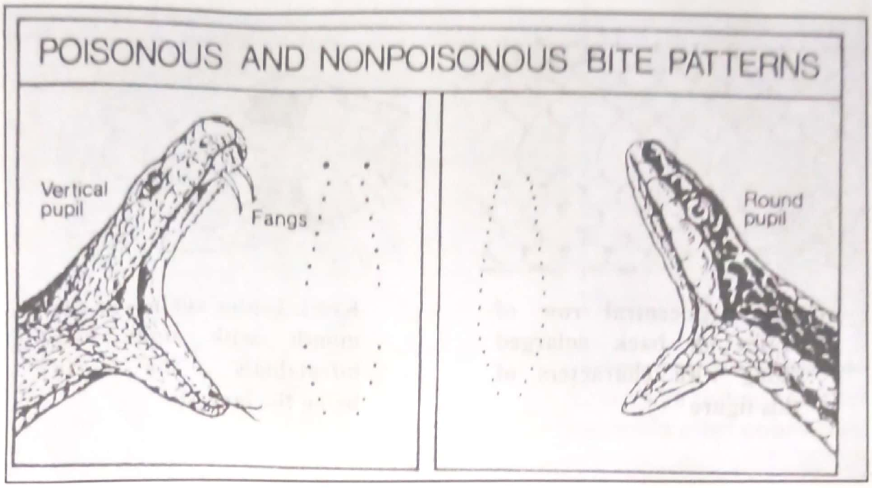
N.B:

1. The snake poison is not effective in it's dry form if taken by mouth.
2. While antivenin is effective even when given after a delay, it is quite common and fatal to have anaphylactic reaction. It should therefore be given only if signs of systemic poisoning appear e.g. ptosis or haemorrhagic signs develope after snake bite.
3. If it is given at the site of bite within few minutes, it prevents local necrosis.
4. It is important to give a test dose prior to therapeutic use to test for serum sensitivity.
5. The antivenin can neutralize the circulating toxin only and not the toxin fixed in the tissue. The toxin's action in the tissue can be antagonized by:
   - a) Neostigmine-atropine therapy in elapid bite.
   - b) Heparin + supportive fibrinogen transfusion in viper, in the following schedule:
     - The patient with systemic elapid poisoning:
       Give initially 5 injections, 0.5mg neostigmine I.V at half hourly intervals followed by similar doses at

intervals of 2-10 hours depending on response, each injection is preceded by 0.6mg atropine sulphate to counteract the bradycardic effect of neostigmine. This is very effective treatment in elapid bites except the cardiotoxic type of cobra which is very rare.

- The patient with haemorrhagic manifestations from viper receives I.V heparin in a dose of 15.000 units followed by 5000-10000 units every six hours +300-600gms of fibrinogen is given in addition.

This type of therapy is found useful not only for fatalities, but as well preventing complications of systemic intravascular thrombosis such as acute renal failure brought about by the toxin induced consumptive fibrino-coagulopathy.

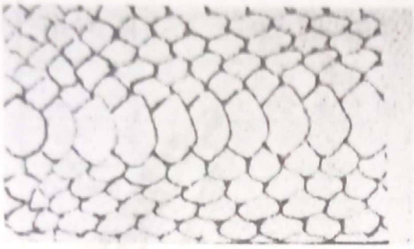


Comparison of the poisonous (pit viper) and the non-poisonous snake bite

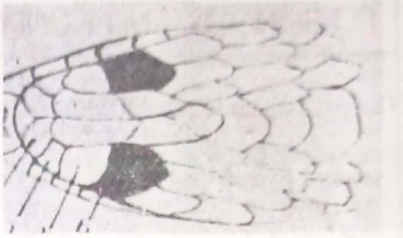
Banded krait

Cobra

Viper Snakes



Kraits , central row of scales on back enlarged along with characters of this figure

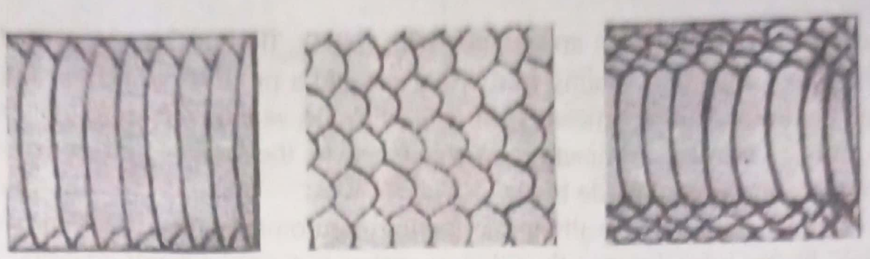


Krait. Under surface of the mouth with only four infralabials , the fourth being the largest

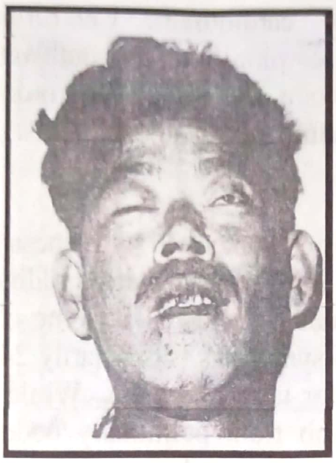
Elapid fangs (cobra in this case) being covered by mucous membrane, are difficult to see.

Viper fangs are easy to see in the erected position as in this case.

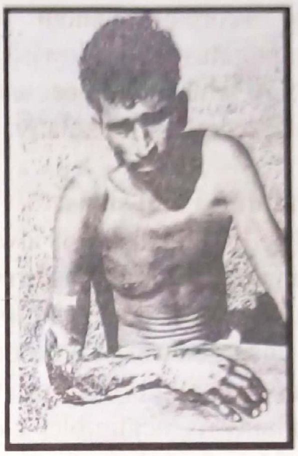
Belly scales of poisonous and non-poisonous snakes



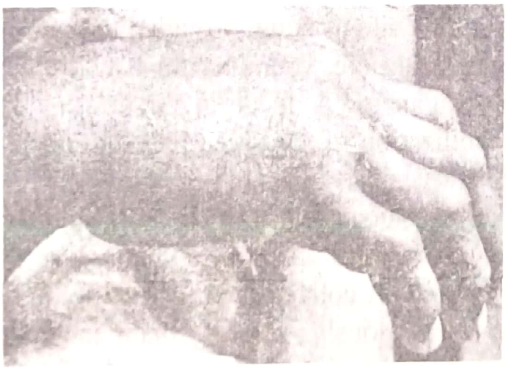
Poisonous **Non-poisonous** **Mixed**



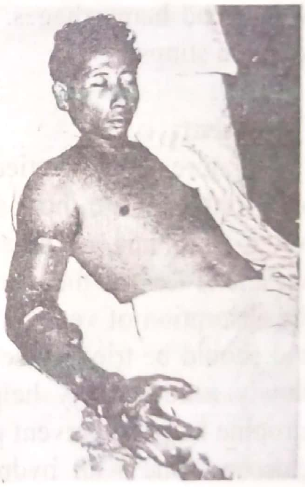
This person was bitten on the hand by an elapid snake (cobra) ptosis indicates systemic elapid poisoning.



Extensive but superficial necrosis after cobra bite



This patient was bitten by a viper on the hand (swelling of the hand is seen)



The patient is in a state of shock from systemic poisoning after viper bite. Serous and serosanguinous blisters are seen on the hand

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[1] Reference: Parik's Textbook of Medical Jurisprudence and Toxicology. C.K. Parik.

SCORPIONS

Scorpions have crab-like appearance with a long, fleshy, five segmented, tail like post abdomen, ending in a broad sac and a prominent hollow sting which communicates by means of a duct with the venom secreting glands. The sting is similar in function to the fangs in the snakes and conveys venom into the wound made by it.

The venom contains toxalbumins having neurotoxins and haemotoxic actions. It's toxicity is worse than that of snakes but only a small quantity is injected.

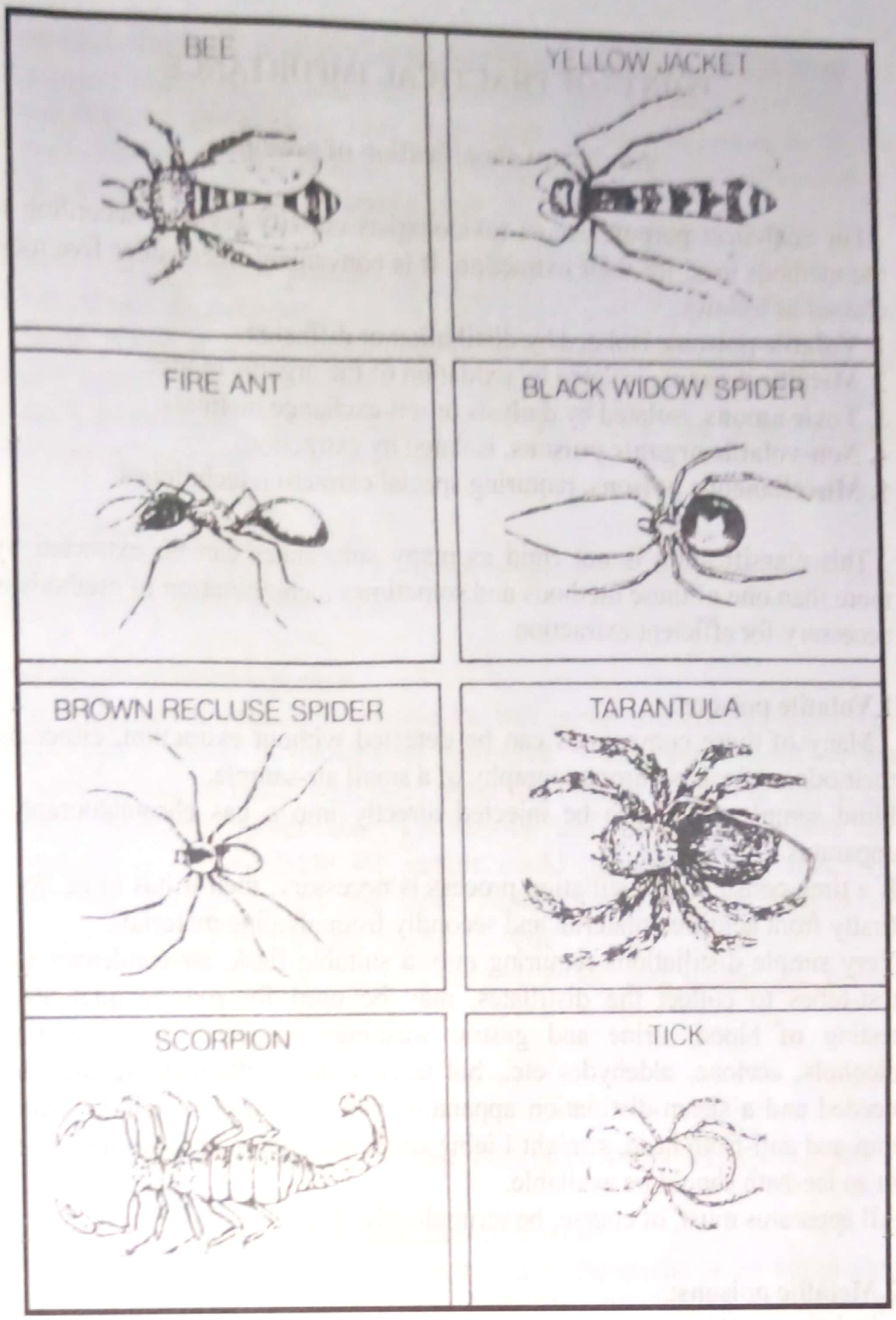
Red scorpion venom contains a potent cardiotoxin. Centruroides sculpturatus is a yellowish, 1-3 inches. Long scorpion found in southwetren U.S.A which produce sever systemic symptoms due to it's neurotoxins. Most scorpions are relatively harmless, producing only local symptoms.

Symptoms and signs:

The local irritation is always very sever. It is characterized by redness and burning pain radiating from the site. There may be headache, giddiness, nausea, profuse perspiration, and muscular cramps followed in some cases by unconsciousness. Although the duration of symptoms is ordinarily 24-48 hours, neuralgic manifestations may persist for upto one week. While the mortality is negligible, children may succumb from pulmonary oedema. Rapid progression of symptoms in the first two to four hours after a sting indicates poor outcome. Pathologic findings in cases of death are widespread hemorrhages. Myocardial damage is found in deaths from red scorpion stings.

Treatment:

A ligature should be tied proximal to the site of sting, provided the bite is on a limb, the site incised, if necessary, and the wound washed with plain water, ammonia or potassium permanganate. A local infiltration of an anesthetic lessens the pain and immobilization of the bitten part diminishes the absorption of venom. A specific antivenin is available for most species and should be tried. Injection of calcium gluconate, 10 ml of 10% solution slowly intravenously helps to relieve muscular cramps and injection of atropine helps to prevent pulmonary edema. A collapsed patient will require glucose-saline with hydrocortisone intravenously. Cardiac manifestations from red scorpion sting will require appropriate treatment. Spraying of 10% DDT kills the scorpions.


BEE
YELLOW JACKET
FIRE ANT
BLACK WIDOW SPIDER
BROWN RECLUSE SPIDER
TARANTULA
SCORPION
TICK

Common venomous insects